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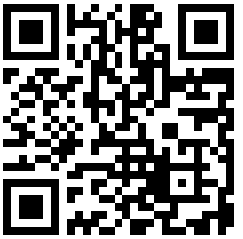

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THE

CLINICAL JOURNAL

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW

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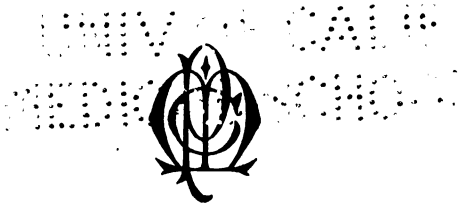

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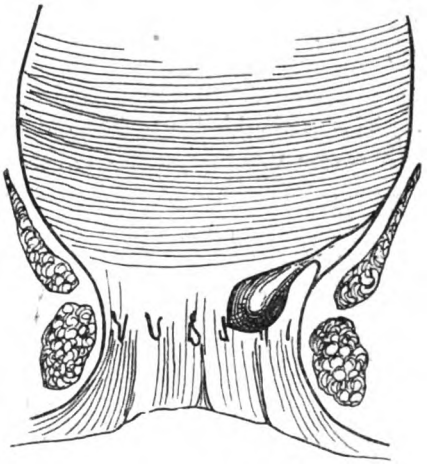
WITH MR. F. C. WALLIS AT CHARING CROSS HOSPITAL.

NEOPLASMS OF THE LARGE INTESTINE AND RECTUM.

GENTLEMEN,—The INNOCENT GROWTHS of the large bowel and rectum may be said to exist in every form, but I wish only to deal with those conditions which most commonly arise, and of these by far the most frequent is the pedunculated polypus.

FIG. 1.



Polypi may occur anywhere in the intestinal canal, but the rectum is undoubtedly the most common situation for polypi which give rise to symptoms calling for treatment. They occur in children and in adults and are usually single, but two or three may be present, or the polypi may be multiple and extend over the whole of the large bowel and into the small intestine; this latter condition I will discuss later.

Whatever the growth of a polypus—*i. e.* whether it is fibroma, adenoma, or lipoma—the common characteristics are that it is a round, smooth swelling attached to a pedicle which may be any leng-

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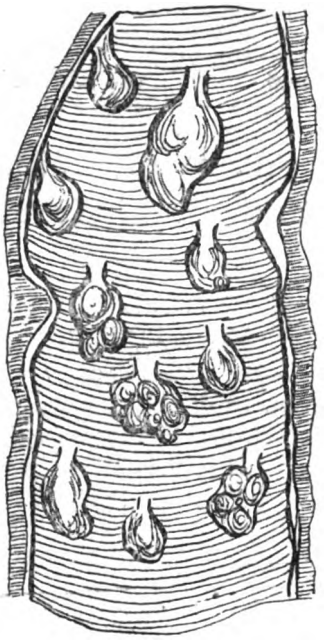
from half an inch to three or four inches, and its usual situation is within the first two inches of the

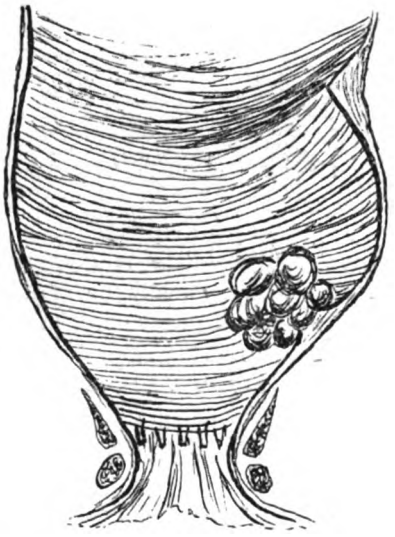
FIG. 2.



bowel. This is the kind of polypus which most frequently comes to our notice (see Fig 1).

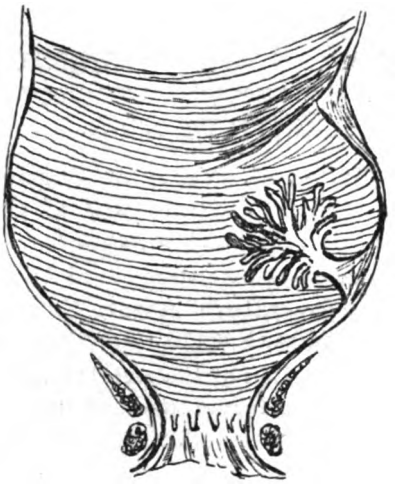
In the rectum these polypi may exist for years

FIG. 3.



ithout causing any symptoms until by the daily :tion of the bowels the pedicle becomes sufficiently ng to enable the polypus to be grasped by the sphincters. When this occurs there is a great deal of straining and spasm of the sphincters, until the polypus is either returned to the bowel or other-

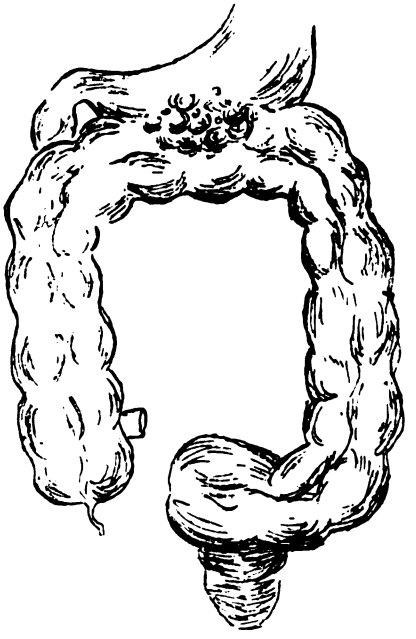
FIG. 4.



wise dealt with. If a polypus is gripped by the sphincters long enough it becomes gangrenous and sloughs off, and thus a cure is obtained.

When a polypus causes symptoms which necessi-

FIG. 6.



tate its removal, there are two methods of doing this, but first of all the rectum should be carefully explored with the finger, and the size of the polypus

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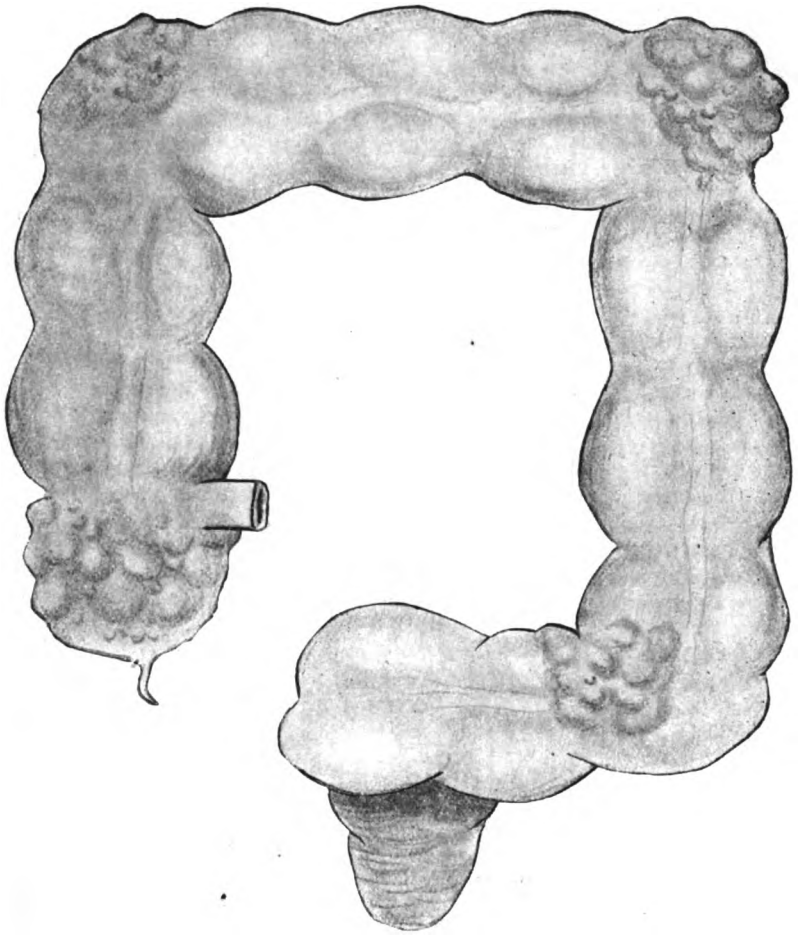
made out, together with the length and thickness and point of attachment of the pedicle. Usually the finger can be hooked round the tumour, which is then by gentle traction brought into view outside the sphincters, a ligature is passed round the pedicle, and the polypus is cut off. No dressing is required, nor is any internal application necessary.

When the polypus has been attached high up and the pedicle is somewhat broad at the base, an anæsthetic should always be given and the sphincters thoroughly stretched before attempting its removal.

In certain rare cases it has been found that a peritoneal pouch is included in the pedicle. It is as well to bear this in mind when dealing with a polypus with a high pedicle attachment, and to take care that the ligature is well tied, with sufficient pedicle in front of it to avoid any chance of slipping.

Multiple polypi are usually adenomatous in structure, occurring in the rectum and over the whole extent of the large bowel. They sometimes exist even in the small intestine. They are most numerous at those places in the large bowel where the intestinal contents are apt to accumulate. The mucous membrane of the bowel shows generally signs of chronic inflammation in cases of multiple polypi.

FIG. 5.



Symptoms. — Hæmorrhage is the symptom for which patients seek relief in these cases, and tenesmus, pain, and diarrhœa are present in a greater or less degree, dependent on the amount of co-existing colitis. As is common in all chronic intestinal troubles, the patients are usually anæmic and debilitated.

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Diagnosis.—If the most prominent symptom in these cases is hæmorrhage a careful examination of the rectum should be at once made. If diarrhœa is the main symptom, and ordinary treatment does not allay it, a local examination of the rectum and sigmoid should be carried out by means of the finger and sigmoidoscope, when some such condition as is shown in the diagram will be seen (see Fig. 2).

Treatment.—As may be imagined, no form of treatment, dietetic or medicinal, is in any way satisfactory, and operative treatment to be useful must be thoroughly carried out.

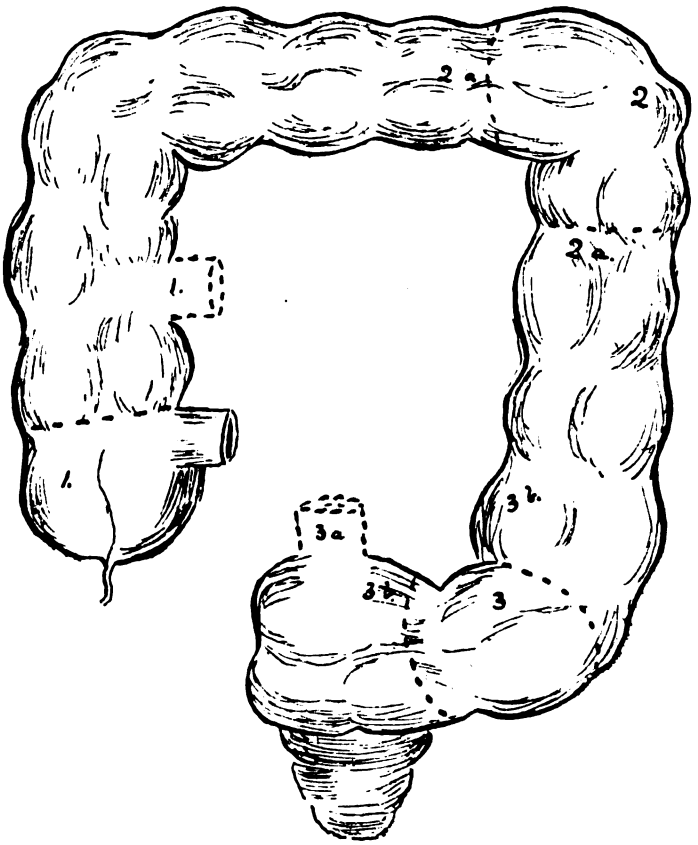
Food should be non-irritating and easily digested.

The drug most useful is opium, which should be given in fairly large doses; this alleviates the pain and lessens the diarrhœa. But whatever palliative measures are adopted they will in no way affect the pathological condition of the bowel.

The benefit of any surgical operation will depend on the limitations of the growths. If they are confined merely to the rectum and lower part of the sigmoid, they can be removed by the electric thermo-cautery through the sigmoidoscope. A long fine hook steadies the growth, and the platinum cautery at a dull red heat sears through the pedicle. The tumour when separated is pulled out by the hook. In this way the various polypi can be fairly easily removed, and there is little or no hæmorrhage.

If the condition pervades the whole of the large bowel, the following treatment may be adopted: The growths in the rectum and sigmoid having been removed in the manner just described, ten days later the abdomen is opened in the mid-line and an anastomosis is made between the ileum and sigmoid. By these means the affected colon is cut off from the contents of the intestines, and the symptoms of hæmorrhage and diarrhœa either disappear or improve considerably.

FIG. 7.



A remarkable case is recorded by Lilienthal in which, after having performed the operation just mentioned, the whole of the colon was successfully removed, the patient making a perfect recovery.

Solitary polypi in the small or large intestine are occasionally the starting-point for an intussusception, and, in fact, most cases of chronic intussuscep-

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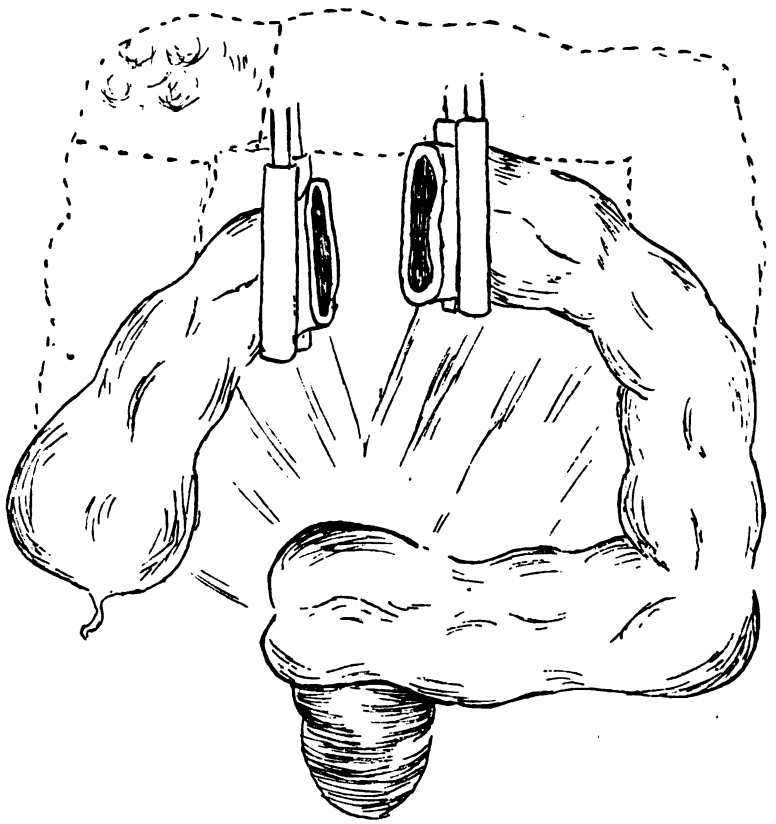
tion are produced in this manner. Bearing this in mind, it is quite reasonable to suggest that certain obscure cases of abdominal pain, associated with a definite swelling which disappears when the pain goes, and recurs in the same area when the pain recurs, may be due to some solitary polypus producing the irritation. I have no evidence in support of this, but it would seem a reasonable deduction under such circumstances as I have mentioned.

Single sessile adenomata grow occasionally in the rectum, and when symptoms occur which entail a readily and somewhat freely. They arise from the mucous membrane of the bowel, and are attached with rather a broad base. They may grow to a considerable size, and often become malignant (Fig. 4).

The symptoms are a copious discharge of mucus and hæmorrhage of a variable character, sometimes oozing, at others loss of large quantities of blood, a feeling of weight and fulness in the bowel, which is often associated with tenesmus.

The aspect of patients suffering from villous growth in the rectum varies considerably. Some

FIG. 8.



rectal examination and recognition of the tumour, this should be removed together with an elliptical piece of the mucous membrane around it (Fig. 3).

It is a matter of considerable importance that the tumour and its base, together with a margin of healthy mucous membrane all round it, should be removed, as this form of tumour is apt to become malignant, and if imperfectly removed a recurrence in a malignant form may grow from the base.

Villous tumours occur in the rectum and are of much the same character as those that occur in the bladder and kidney. They consist of spongy masses with long villous processes which bleed appear in the very best of health, and so, indeed, they are but for this. Others become anæmic and lose weight; this latter condition is brought about by the loss of blood they sustain.

On examination the tumour feels soft and velvet-like, and the various lobules can be traced out with the finger. The mucous membrane can be felt to run up to the base of the tumour, which spreads out immediately above this—a totally different condition to carcinoma, as far as digital examination is concerned. These tumours are rare, but with the definite points given above may be always recognised.

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Treatment.—The treatment of these cases must not be one of any half measures.

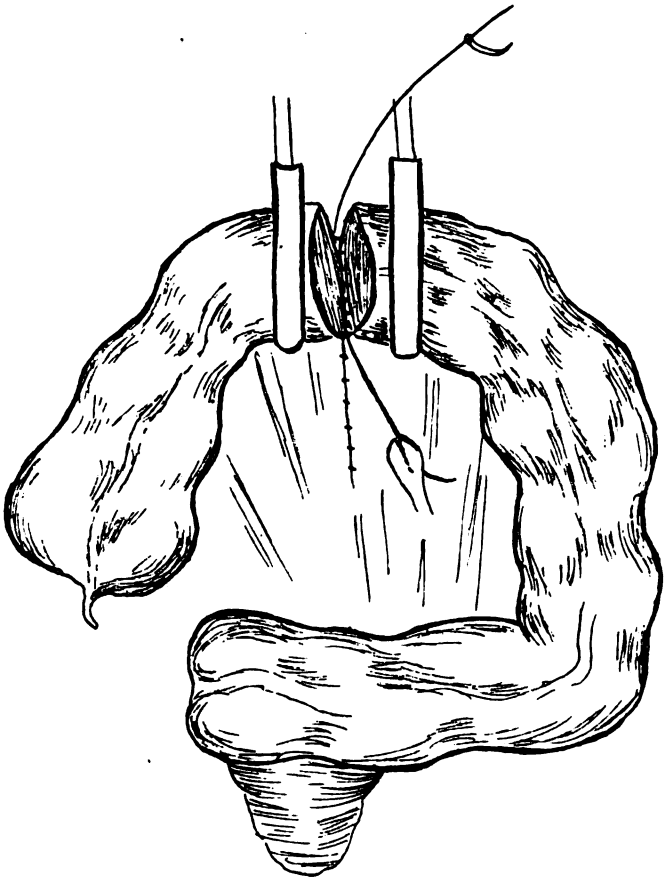
When local removal has been performed on the basis that the clinical features of the disease point to an innocent character the growth has almost invariably returned after a short interval in a malignant form, with wide-spreading infiltration of the surrounding mucous membrane. Such being the case, it is far better surgery to act on the same lines as one does in dealing with malignant growths.

usually occur, giving the symptoms which will lead to the diagnosis, and then discuss the various operations which may be undertaken for the cure or relief of this dire disease.

Cancer of the large bowel may occur in any part of it, but it more frequently occurs at those situations where fæcal accumulations are apt to occur, viz. at the cæcum, the hepatic, splenic, and sigmoid flexures of the colon, as shown in the diagram, Fig. 5.

It may also occur as an extension from some

Fig. 9.



The various operations for these I will describe later.

The MALIGNANT TUMOURS of the large intestine and rectum which I now propose to discuss briefly are those which grow from the mucous membrane, and consequently are carcinomatous in one form or another.

Adenoid cancer is the most frequent of the various forms, but medullary, scirrhous, colloid, and epithelial cancer also occur, the latter most frequently at the anal margin.

All I can do in the time allotted to me is to indicate the situations in which these growths

neighbouring viscus, as shown in Fig 6, where a growth originating in the stomach has invaded the transverse colon. (See Fig. 6, p. 2.)

The symptoms in these cases are constipation alternating with diarrhœa, abdominal pain, loss of weight, loss of blood rarely, and a tumour in one of the situations already indicated.

Dependent on the situation and size of the tumour, there may be partial intestinal obstruction, which will be shown by the distended condition of the abdomen, associated with general malaise, occasional vomiting, a foul tongue, and often a rise of temperature. This partial obstruction may

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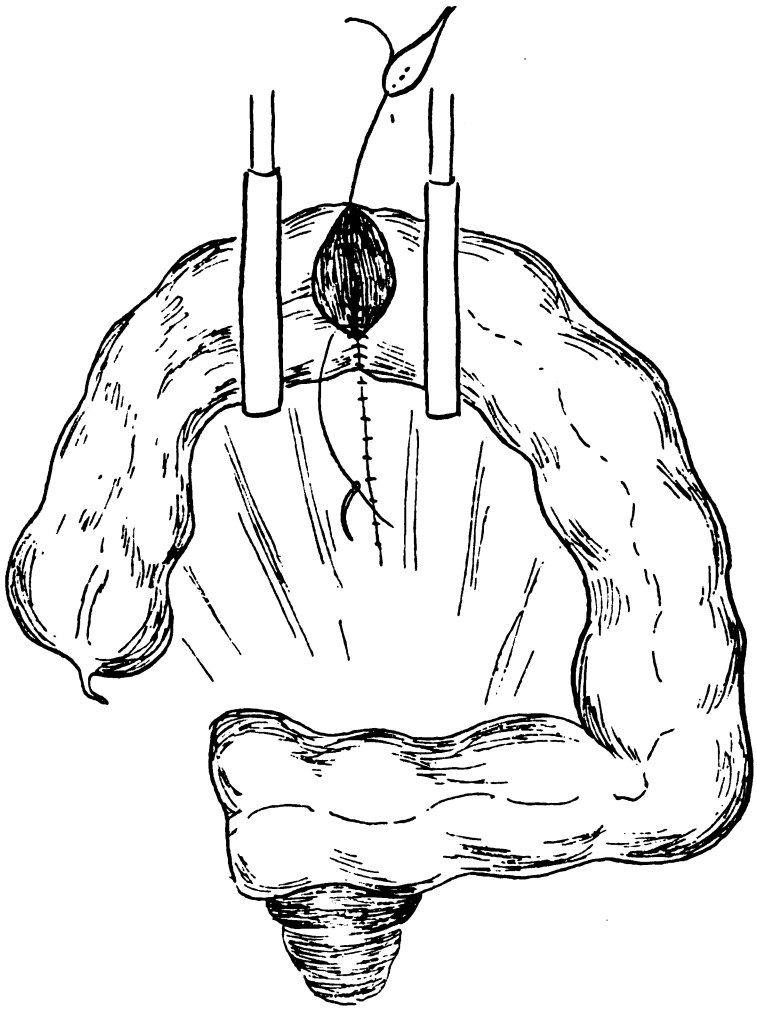
at any time become complete. In all these cases a careful examination of the abdomen is of the greatest importance, and if from any cause, such as obesity, tenderness, or nervousness, the abdominal walls are not sufficiently relaxed, an anæsthetic should be given. Indeed, it is almost always more satisfactory to do this before expressing any opinion as to diagnosis or treatment.

Growths which appear to be firmly fixed when examined without an anæsthetic become freely movable when examined under an anæsthetic. The hardness and nodular outline of these swellings can also more easily be made out, thus differentiating them from fæcal accumulations, which are rounded, smooth, and of a doughy consistency.

Fig. 7 is intended to show some of the methods which may be adopted when growths occur at 1, 2, or 3.

If a cæcal growth is removable, then the ileum is transplanted into the colon higher up. If the growth is at 2 or 3 and removable, the growth is removed, and the two cut surfaces 2 *a* and 2 *a*, or 3 *b* and 3 *b* are brought into apposition and sewn together. If the growth is too widespread and infiltrated to allow of its being removed, then the end of the ileum is transplanted into the lower end of the sigmoid, 3 *a*, thus throwing nearly the whole of the large intestine out of work. It is this latter operation which has to be undertaken when the colon is affected with multiple polypi.

FIG. 10.



The next figures (Figs. 8, 9, 10, and 11) show the method by means of which an end-to-end suture of the large bowel is brought about.

In Fig. 8 the dotted line shows the original situation of the bowel, and below this are the two cut edges about to be apposed after removal of the tumour.

The method of sewing the bowel together is shown in the next three figures.

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Fig. 9 shows the first line of sutures, which, beginning at the cut edges of the mesocolon, sews together by a continuous suture the posterior edges —going through the whole thickness of the gut.

Fig. 10 shows the second row, a repetition of the first in that all the coats of the bowel are taken in, the difference being that in the first row the suture goes from the mucous membrane to the mucous membrane. In the second row the suture goes from serous coat to serous coat. Great care must be taken that the two ends shall be thoroughly closed, and that the cut edges of the mesocolon are approximated by a few interrupted sutures.

Fig. 11 shows the completion of the first line of sutures, and the dotted lines indicate the situation of the second row, which goes right round the bowel, but this suture takes up the serous and muscular coat only, and does not touch the mucous coat. The small diagram underneath is intended to show the doubling in of the bowel which is effected by the second row of sutures, which is produced by the close approximation of an appreciable amount of the serous coat, and effectively prevents any leakage.

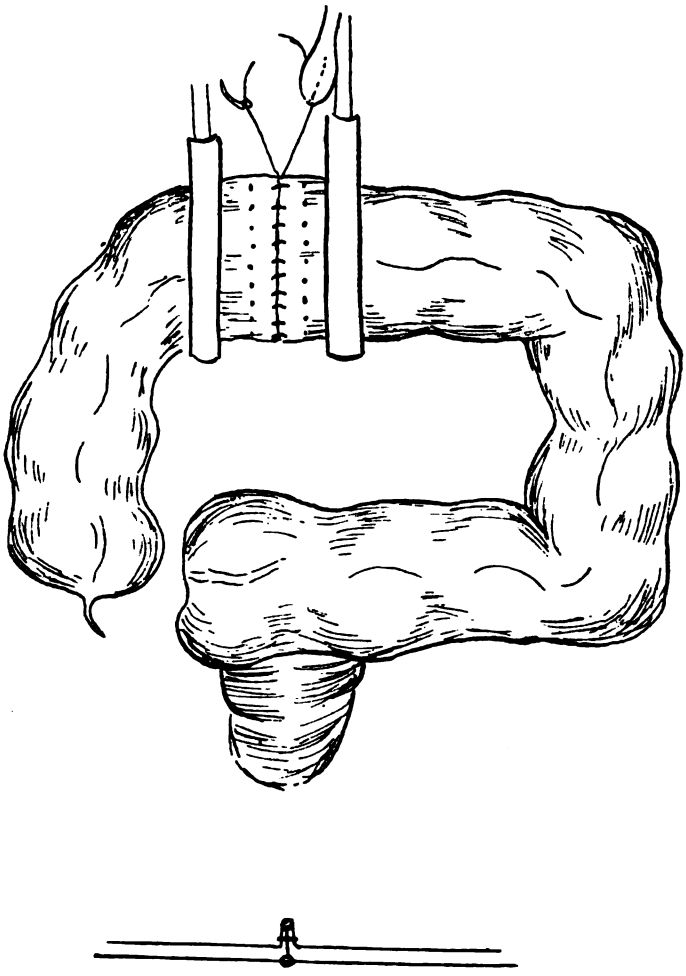
The next three figures show the plan of making a lateral anastomosis of the bowel.

Fig. 12 shows the first line of continuous sutures which approximates the two serous coats, taking up the serous and muscular coats only and the incisions into the lumen of the bowel on either side.

Fig. 13 shows the approximation of the two inner edges of the incised bowel.

Fig. 14 shows the sewing of the two outer cut edges half completed. The four small lines on either side indicate where the fourth row of sutures would come, and when this row is finished the last stitch is tied to the first of the first row of sutures, thus completing a double row of sutures and making a most effective union.

Fig. 11.



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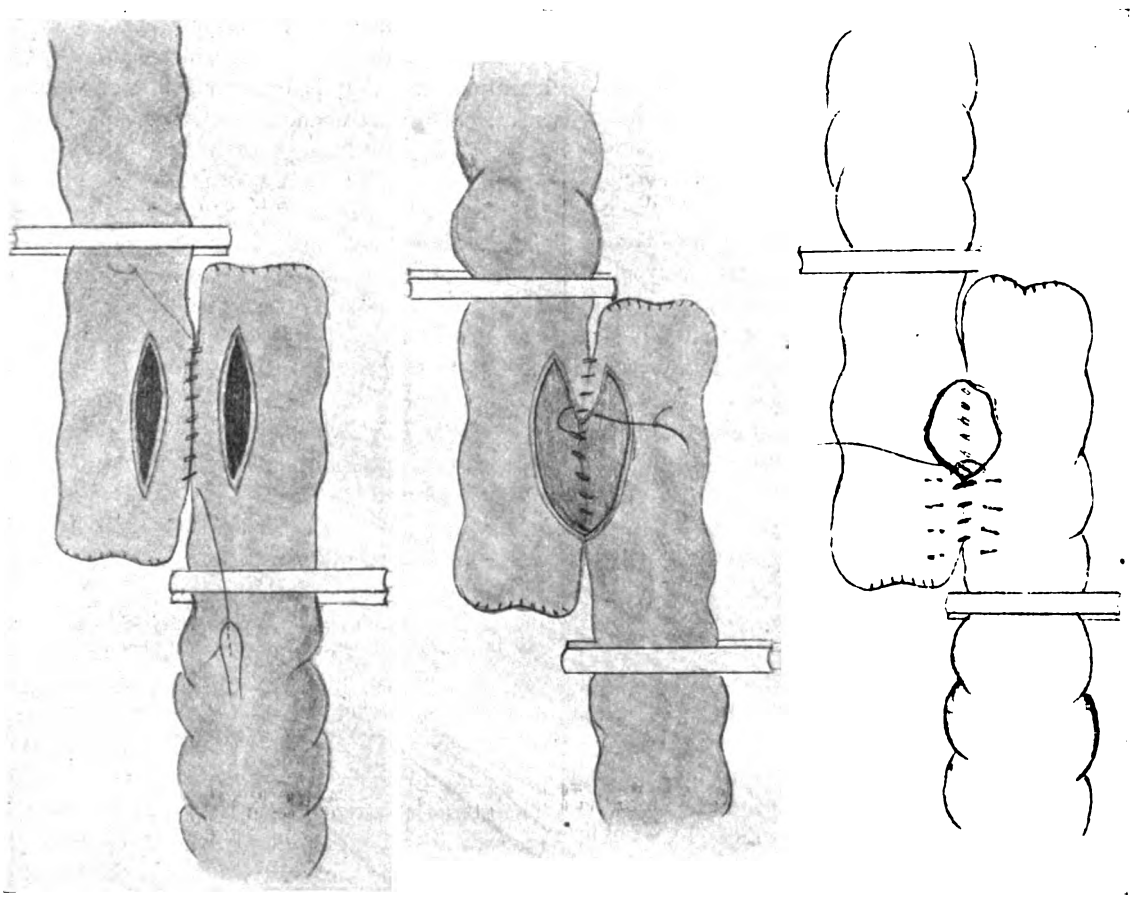
Comparing the two methods, there is no doubt but that greater secusity is obtained by the lateral anastomosis, but it takes somewhat longer to do, and in addition the two cut ends of the intestine have to be inverted and oversewn.

I am convinced that Murphy's buttons are not desirable for use in the large bowel. As long as the button is *in situ* the lumen of even the biggest is not large enough to allow accumulated and possibly hardened fæces to pass through, and the consequence of this is that the bowel separates

FIG. 12. FIG. 13 FIG. 14.



and a fatal result obtains, as was my experience in the following case: A patient with marked pyloric obstruction came under my care. When the abdomen was opened it was found that a carcinomatous mass involved not only the pylorus but also had invaded the transverse colon to such an extent that the large bowel was at this point almost obstructed. A posterior gastro-jejunostomy was performed, and then the ascending colon was brought across and fixed to the descending colon by means of the large-sized Murphy's button. The patient did well until the fifth day, when peritonitis supervened and death occurred, the next day. At the post mortem it was found that the solid accumulation in the descending colon was unable to get through the lumen of the button, and the button, being dragged on, had separated the apposed peritoneal surfaces of the colon and leakage had occurred. The gastro-enterostomy sewing was quite sound and water-tight.

Even if the result is successful there is considerable difficulty in passing the button. The following case is an example of this: A patient under my care had partial intestinal obstruction. When the abdomen was opened a carcinomatous mass was seen to occupy the transverse colon, the growth was fixed, and there were many glands involved. The growth could not be resected and the ascending colon was attached to the sigmoid by means of a large Murphy's button. The patient did well, but the button had to be extracted from the rectum three weeks later, the size of the button being such that the patient was unable to get rid of it in the ordinary way.

April 16th, 190€.

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A LECTURE

ON

THE OCCURRENCE OF ABORTION AND PREMATURE LABOUR.*

By F. J. McCANN, M.D.Edin., F.R.C.S.Eng., M.R.C.P.Lond.

Physician to In-patients, Samaritan Free Hospital for Women.

GENTLEMEN,—I have here a specimen of a complete ovum (fœtus, placenta, and membranes) from a case of premature labour. This specimen was kindly provided for me by one of my audience this afternoon, and after the lecture I will demonstrate the various parts of this specimen.

I thought I could not do better than say something in this lecture on a subject which is very common, so common as to make it of very great importance indeed, and that is the occurrence of abortion and premature labour. I shall not say anything about the causes of abortion, because they are so well stated in the books. But I may say at once that the chief causes of abortion in the early months are abnormal conditions of the endometrium and uterine displacement, and in the later months the chief causes are syphilis and albuminuria. When you are called to a patient who is supposed to be aborting you should determine—firstly, whether she is pregnant; secondly, whether the pregnancy is intra-uterine or extra-uterine; and thirdly, whether the abortion is a threatened abortion or an inevitable one First of all, with regard to the question whether she is pregnant or not. In the later months the diagnosis may not be very difficult, but in the early months it may be. The signs you should chiefly rely upon are these: first of all, the enlargement of the uterus corresponding to the period of menstrual suppression. In some cases of abortion you will get no history of amenorrhœa; a patient may have had a menstrual period twice or even three times and still be pregnant. But in these cases you re y upon the uterine enlargement, and the altered consistence of the uterus. The cervix becomes softened, and so also does the body of the uterus. Now, with regard to estimating the consistence of the uterus during pregnancy, it is necessary to take every opportunity of noting this physical sign until you have obtained a clear idea of the alteration produced. Once you have that firmly fixed in your minds, it will be a great help to you in the diagnosis of pregnancy. You have to contrast the consistence of the pregnant uterus with that of the unimpregnated uterus and with the uterus which is the seat of chronic metritis. It is important, then, to know the exact amount of softness which you feel in a pregnant uterus, and how you can indent it, and how that indentation readily disappears. There is another important sign in early pregnancy which is not often described, but which is worthy of attention, and that is transverse uterine enlargement. The first change which takes place in a

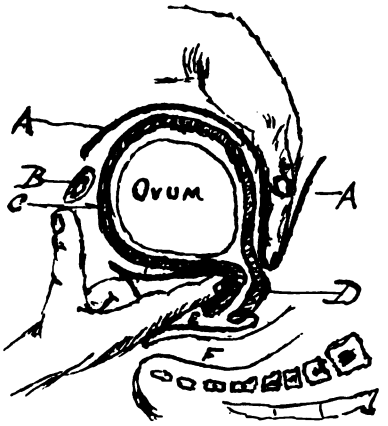

Diagram of a median section of the pelvis to show the method of obtaining Hegar's sign. The lower uterine segment is compressed between the hand on the abdominal wall and the fingers in the vagina. AA. Anterior abdominal wall. B. Symphysis pubis in section. C. Uterine wall with contained ovum. D. Cervix uteri. E. Vagina. F. Rectum.

pregnant uterus is an enlargement transversely. The uterus is flattened from before backwards and transversely enlarged, and if you find this transverse enlargement it is a sign upon which a good deal of reliance may be placed. The next change is an antero-posterior enlargement. And when you have transverse enlargement plus antero-posterior enlargement, the result is a globular enlargement, which you find about the third month. At that period, or even as early as the sixth week, you get an important sign, which is known as Hegar's sign (see diagram). On bimanual examination with two fingers in the vagina and the other hand on the abdominal wall, a soft, compressible area is felt between the globular uterine body and the cervix,

* Delivered at the Polyclinic.

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so soft indeed in certain cases that the fingers of the external hand almost meet those in the vagina at this area. It is supposed that the portion of the ovum occupying the lower uterine segment is forced into the upper part of the body of the uterus and that the empty and softened lower uterine segment can be readily compressed between the fingers. Once you have made out Hegar's sign in one or two cases, and appreciated what it is, you will find it one of the most valuable signs of pregnancy, especially when you are misled by the history, as not infrequently happens. In addition, you must examine the breasts, but unfortunately, in the early months, the breast changes do not help you very much. If breast signs are present you will give due weight to those signs, but if they are absent that is no proof against the existence of pregnancy. Breast signs are more likely to help you in a first pregnancy than in any subsequent pregnancies.

Having settled that the patient is pregnant, the next point to determine is, Is the pregnancy intra-uterine or extra-uterine? You might be astonished to hear it, but this does not seem to occur to many men, that when they are called to what is regarded as a threatened miscarriage they may be going to a case of tubal fœtation. And I can tell you of an instance where a patient who was complaining of a good deal of hæmorrhage was seen by a medical man who had all preparations made for curetting the uterus; another medical man was consulted, who sent her into the hospital. It was found she had a large retro-uterine hæmatocele. If the doctor had curetted the uterus the results would have been disastrous, not only to his own reputation, but probably also to the life of the patient. When you are called to a threatened miscarriage you should determine whether that pregnancy is intra-uterine or extra-uterine. You know that the two are very similar as regards their history. You may have a patient telling you that she has missed two periods, or such a statement may be absent from the history, and that she has been attacked with pain and free blood loss. You know that hæmorrhage from the uterus occurs in tubal pregnancy when a decidua is passed, either in part or in its entirety. These cases are often seen by those who do hotel practice, and if you are summoned to what you suppose to be a miscarriage you ought to determine the situation of the pregnancy, and to do that you should carefully investigate the condition of the broad ligaments, and feel if there be any swelling outside the uterus. If the pregnancy is extra-uterine, prompt surgical treatment will be indicated in the majority of the cases.

Next you have to settle whether you have to deal with an abortion which is threatened or one which is inevitable. These two terms are so frequently used in text-books that one must adhere to them. An abortion is threatened when there is pain or when there is hæmorrhage. There may be pain of considerable severity, and yet the trouble may pass off. The patient may have hæmorrhage of moderate severity without mishap, but she cannot have a severe or long-continued hæmorrhage without the abortion becoming inevitable. If there be pain and hæmorrhage together, and if, on examination, you find the ovum protruding at the os, abortion is inevitable, and the sooner it is terminated the better for the patient. You may meet with many patients who are very anxious that abortion should, if possible, be prevented and the pregnancy go on uninterruptedly. The question is, What treatment should be adopted? The first and most important precaution is to keep the patient at rest. Rest in the horizontal position is the best treatment for all uterine hæmorrhage, whether associated with pregnancy or not. The next is to give a dose of opium, and the best preparation is either the Liquor opii sedativus (Battley), which, by the way, is far superior to the officinal preparation, or nepenthe. Personally, I much prefer the first of these. A patient I recently attended was very anxious that her pregnancy should go on uninterruptedly, and when I examined her I found the ovum protruding. I gave a large dose of Liquor opii sedativus, which produced sleep, and the ovum returned into the uterus. It was most remarkable to see the effect of opium in quieting the uterine contraction in that case. Ultimately, however, that uterus had to be emptied of its contents. As you cannot go on giving opium indefinitely, you must prescribe something else. What you should give later on to the patient is bromide of potassium. That is the best drug to administer for hæmorrhage during pregnancy, and it is well to give it in aromatic spirits of ammonia—10 gr. of bromide of potassium and 20 ♏ of aromatic spirit of ammonia in half an ounce of water three times a day. I know some authorities recommend ergot in small doses, but personally, I cannot convince myself that it is of

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any use in these conditions. Bromide of potassium is certainly useful; it has proved its worth over and over again. Another drug which you may give is hydrastis, Tincture hydrastis canadensis, in 20-m doses every four hours. There is another drug of which a great deal is heard, and that is extract of viburnum primifolium, given in 1- or 2- or 3-drachm doses three or four times a day. This is a drug which came from America with a great reputation, but in my hands I am bound to say it has not yielded good results. It may be that I have not obtained good preparations, for, as is well known, these extracts are sometimes unsatisfactory.

Another most important precaution in such cases is to keep the bowels acting regularly. There is no doubt that nothing can beat castor oil for this purpose; although it is disagreeable to take, yet it is very efficacious. There is one thing you should not recommend, and that is, giving an enema in these cases. An enema, especially if it is badly given, or if it acts very freely, is one of the best methods of hastening abortion.

To patients with threatened abortion you may give another drug, namely chlorate of potassium, which was recommended by the late Sir James Simpson, on the assumption that it gave off sufficient oxygen to assist in prolonging the life of the fœtus. In the chemical laboratory chlorate of potassium is used for the production of oxygen, but whether that same beautiful experiment occurs when you give it internally I do not know. But whatever the explanation be, it seems to act very well in certain cases. Sir James Simpson's directions for giving it are to administer 20-grain doses three times daily on an empty stomach, and it may be pleasantly taken in aerated water. He used to say that the dose usually given was too small; 5 to 10 gr. does not seem to be sufficient, you need to give 20 gr. Another drug which certainly helps patients who threaten to abort is iodide of potassium. I have more faith in iodide of potassium in preventing miscarriage than in almost any other drug, because I have no doubt a large number of the cases are syphilitic in origin. But please do not go away with the idea that all these cases are syphilitic. I believe that quite a large group of cases are also due to placental disease, probably obliterative endarteritis, not syphilitic in origin, and that condition is benefited by iodide of potassium. I have, I think, proved this in several of my private patients, by going into the matter and really sifting the history as well as one can, although, as I have already said, histories, especially where syphilis is concerned, are not very trustworthy. Still, there are one or two cases where, from the evidence I have been able to obtain, syphilis can be excluded. So much for the treatment of the threatened cases.

Now with regard to inevitable abortions. What is to be done for them? Supposing you are called to a case where an abortion is inevitable, where there has been free hæmorrhage and the ovum is protruding, what is to be done? You, of course, cannot wait there all night, and you must do something. The best treatment is to give the patient 30m of the liquid extract of ergot, and order that to be repeated every four hours if necessary; then take a piece of iodoform gauze (which you should have in your bag), at least one and a half yards in length, and four inches broad, and having first of all cleaned the external genitals with soap and water, and if necessary cutting away some of the vulvar hairs, which are frequently matted with blood and discharge, swab the external genitals over with 1 in 2000 corrosive sublimate, and the vagina with a similar solution, expose the cervix with a speculum or retract the posterior vaginal wall with two fingers, and then with a pair of forceps take hold of the end of the gauze and plug it in as tightly as possible into the cervix and leave the remainder in the vagina. There are to be no more pledgets of wool with a string attached, and no more pieces of dirty cotton used—at least, by those who have attended the Polyclinic. You ought to use clean gauze, and then you will never see septic abortions, which are still so frequent in practice. From one patient I removed a dirty pocket-handkerchief which had been employed as a vaginal plug! Use this iodoform gauze, plug it into the cervix, and fill the vagina with it, give a dose of ergot, and then you can go home to bed. When you return next morning, take out the gauze and probably you will find the fœtus will be on top of it. Supposing there is considerable hæmorrhage and the cervix is not sufficiently well dilated, what is to be done? In these cases I believe the best treatment is to use a tent. There are tents *and* tents. If you are going to use a tent, you must use one in the way a surgeon would use it if he were going to insert it

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into the abdominal cavity. That is to say, your tent must be aseptic, and it must be inserted with all precautions which you use for a surgical operation. Unless you feel confident on this point do not use it. The question then arises, What tents will you use? The best are the laminaria tents, and you can make them aseptic by keeping them for at least fourteen days, either in a solution of absolute alcohol or in a solution of iodoform and ether 10 per cent. The latter is the one which I prefer, for the reason that the ether evaporates and leaves a coating of iodoform on the surface of the tent. I may say I am one of those who believe in iodoform. I know it has a horrid smell, and that patients hate the smell of it, but I think that if you point out to them the great advantages it has as regards safety they will put up with it. For wounds about the vagina and rectum there is no doubt iodoform gauze keeps the parts aseptic in a way that no other gauze does. I have used sal alembroth, double cyanide, and other gauzes, but they are not to be compared with iodoform in efficiency. In introducing a tent, then, you take the same precautions in cleansing the vulva as for other operations, for it is from the vulva that most of the infection is carried, especially among the poorer classes. You are called to a miscarriage case and find the vulvar hairs matted together with blood and discharge, and it is really absurd to suppose that unless you clean the external genitals thoroughly you will ever get a case to do well. If you are using a tent, you must cleanse the external genitalia, and having exposed the cervix you may pull it down with a volsellum just to fix it, then introduce the tent into the cervix, using that size which you find from your examination will pass into the cervical canal. The important point is to make sure that you get the tent into the cervical canal, and that its extremity is flush with the external os. To insert a tent and leave half of it out is worse than useless; it must be entirely in the cervical canal, and the end must be flush with the external os. Having got it in, you keep it there with a plug of iodoform gauze, and you should not leave it *in situ* longer than six to eight hours. You then take it out, and frequently after you have taken it out the uterus will be sufficiently dilated to allow the finger to pass in. If it is not you put in another tent and repeat the process. The great advantage of a tent is that it stops hæmorrhage, and it is the best of all dilators for the cervix. Metal dilators, especially the larger sizes, tear the cervix, they do not dilate it. The tent, however, swells, and by its actual presence in the cervical canal stimulates the fundus to contract whilst the cervix dilates; in fact, it causes an imitation of the normal mechanism. Unfortunately, tents were given up because of the complications which arose from using them, simply because practitioners would persist in carrying sponge tents and laminaria tents at the bottom of a dirty bag and put them into the cervix without previous precautions, hoping for a good result. But if you use tents in an intelligent and clean manner, they will yield most excellent results, and there is no method of dilating the cervix to be compared in the matter of efficiency with them. I do not like to be prophetic about anything, but I feel that tents are going to be used much more in the future. Supposing you have a sufficient degree of dilatation, you then proceed to remove the ovum, and in removing the ovum the best instrument is the finger. In doing this let me mention a little practical point, namely that in order to reach the fundus with the finger it is always well to tilt the uterus slightly backwards, so that you get it quite straight. If you press the fundus downwards and forwards, you will increase the difficulty in reaching the top of the uterine cavity with the internal finger. Moreover, in some cases it is advisable to use the middle finger, because it is longer, and with it you may hook out any loose pieces of placenta or membrane. In that way you simply clear out the ovum, and if there are any remnants left behind you may use a curette. And here let me say that the curette should be used with the greatest gentleness; it should be employed rather as a rake than as a scraper. It is very easy in these abortion cases to scrape a hole in the uterus. Some books tell you to go on scraping until you get down to firm tissue. If you do that the firmest tissue which you will get down to will be the pelvic bones! And following that advice, I have, in my experience, seen several cases in which a hole has been made in the uterus, simply because men forget that the uterus is extremely soft, and it is easy, not only to scrape a hole in it, but to push a curette through it. A strong man with a curette in his hand can do a great deal of damage. You should, in such cases, use a blunt curette, and use it with great gentleness.

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Having cleared out the uterus in that way, the next step is to sponge out the cavity with 1 in 3000 corrosive sublimate. I am a great believer in corrosive sublimate, whatever may be said about it on theoretical grounds. I believe it does its work well. In sponging out the uterus you will find squares of gauze very useful, about two inches square, wound round a pair of long uterine forceps. Every man in practice ought to procure a pair of long forceps; it is an instrument you cannot very well do without. Armed with long forceps, iodoform gauze, a little cotton-wool, and a bottle of corrosive sublimate tablets, you are equipped to attend any case of abortion. If you wind the gauze round these forceps and wipe out the interior of the uterus, you will find it very efficacious. I believe swabbing to be more efficacious than douching, and, moreover, douching requires an apparatus, and you have either to take one yourself or use one belonging to the patient, and the latter will probably be dirty. These squares of gauze catch in their meshes little pieces of placenta and membrane and clot, and you will be astonished how clean you can get the interior of the uterus by that method.

If there is no very great amount of oozing you need put nothing in the uterus at all. If there be very free oozing and you are afraid to leave it, you can put in a plug of iodoform gauze. You may leave this gauze *in situ* until the next morning, when it should be pulled out with forceps. I strongly advise you not to use your fingers, and to make sure that all your work is carried out in a cleanly manner. The patient is then left alone afterwards. No douching is required. It is so common now to see that patients, after a miscarriage, are douched for eight or nine or ten days. But why should you douche them? I know that I was taught to do that, but after I had had some experience I asked myself, Why? The douche does not go into the uterus; it washes away the little discharge from the vagina, but if the vagina is clean that discharge will run away. The douche, in fact, often introduces a source of danger, especially if it is given in an unclean manner. I am sure many patients are infected afterwards by the douche. It is necessary to keep the external genitals clean by frequent bathing with 1 in 2000 corrosive sublimate and the wearing of an antiseptic diaper.

Now I must say a word about cases which are septic. If the interior of the uterus is infected and you have a sloughing piece of placenta *in utero*, immediate removal is imperative. You must not delay an instant in these cases. And in carrying out the removal of the placenta you will do better to put the patient under an anæsthetic if it is possible to get a colleague to go with you for that purpose. If you do not give an anæsthetic it is very difficult to dilate the uterus with metal dilators, and tents require too long. Make every endeavour where there is septic trouble to give the patient an anæsthetic, and having dilated with one or more of the metal dilators, get your finger in and remove the placenta. For this operation I strongly advise you to use rubber gloves. They only cost half a crown, and if they were generally used I believe they would do much to diminish the rate of morbidity from puerperal sepsis. They protect the doctor's hands and they also protect his other patients. In septic cases I always use rubber gloves; if I did not, I think I should have to give up my occupation. If I had a case of this sort to-night, and had to do an abdominal section next day, I should, except for the gloves, have to forego the second. The gloves can be boiled in a clean pot for three or four minutes and are then ready for use. Remember in all septic cases that the great desideratum is to avoid doing too much injury. If you dilate freely and lacerate the cervix, you will be opening up new channels for infection; therefore use the utmost gentleness and delicacy; clear out the sloughing piece of placenta and sponge out the uterus in the way I have told you. It is quite unnecessary to plug with gauze; in fact, I believe it is harmful, because, the cervix being sufficiently dilated, any discharge will drain away.

I had very much more to say, but the time has gone. I must, however, mention that where repeated abortion occurs the cause may be uterine displacement, and if you replace the uterus and keep it in place with a Hodge pessary for three or four months, the pregnancy may go on uninterruptedly. In other cases you may have to curette the uterus, because frequently it is some abnormal condition of the endometrium, and it is in these cases, above all others, that the iodide of potassium and the chlorate of potassium are of great use.

Lastly, it must be remembered that if continued hæmorrhage occurs after an abortion, especially if it be a hydatid mole, you must always be on the look-out for the possible occurrence of malignant disease—deciduoma malignum or chorion epithelioma. Fifty per cent. of the cases occur after hydatid mole, so if you attend a patient with continued hæmorrhage who has expelled a hydatid mole, the interior of her uterus should be examined at once, and if any new growth is found the uterus should be removed. These cases are relatively rare, but they are of sufficient frequency to lead you to be suspicious if hæmorrhage continues after abortion.

April 16th, 1906.

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A DEMONSTRATION OF THE ACIDITY OF THE URINE FROM A NOVEL STANDPOINT.

At the Medical Graduates' College and Polyclinic.

By LEONARD WILLIAMS, M.D., M.R.C.P.,
Physician to the French Hospital; Assistant Physician to the Metropolitan Hospital.

THE method of examining the urine which I am about to describe is known in France as Joulie's method. It was first expounded in 1901 in a lecture given at the Institute Pasteur* by the distinguished chemist whose name it bears, and it has since been very extensively advocated and practiced by many physicians, notably by Dr. Jean Nicolaidi,† the majority of whose observations have been made at the Hôtel Dieu. My own experience of the method has been limited to a few cases upon which I have tried it in the wards of the French Hospital, and among out-patients at the Metropolitan, but this experience has been sufficient to convince me that it is well worthy of a much wider recognition in this country than it has so far received.

Before entering into the details of the method, it is necessary to explain briefly the principles upon which it is based. First, then, it would seem that we have so far held erroneous views on a very elementary physiological question. We are taught that the blood is alkaline, whereas it is in reality acid. It is admittedly alkaline to test-paper, but there is nothing either final or supernatural about test-paper, and when the evidence which litmus affords is in direct contradiction to demonstrable scientific facts, it is surely unreasonable to allow ourselves to be dominated thereby. The urine is derived from the blood, and the explanation of how the one, an acid fluid, could be derived by a process of filtration from the other, an alkaline fluid, has always presented a problem to the curious. The problem, in effect, has been an imaginary one, because the acid urine is derived from a fluid which, though leaving a blue stain on litmus paper, is nevertheless chemically acid. The acidity of the urine is due to the presence in it of acid phosphates. These phosphates are also present in the blood, but their acidity in the latter medium is masked by the presence of an excess of bicarbonates, which, though chemically acid salts, inasmuch as they are capable of taking up another atom of base, nevertheless give a blue reaction with litmus. These bicarbonates are for the most part excreted, not by the kidneys, but by the lungs and skin, so that they do not appear in the urine, at any rate in sufficient quantities to affect its action on test-paper. The so-called alkalinity of the blood is due then to the presence of bicarbonates, which are themselves acid salts, so that it is clear that whatever may be the verdict of litmus, chemically and practically the blood is an acid fluid. Nor is there any lack of physiological evidence in favour of this view. We know that the phosphates of calcium and magnesium are so sensitive to any suspicion of alkalinity that they readily precipitate themselves, even in a medium which is still faintly acid. We know further that these earthy phosphates are very frequently present in the urine, especially after meals. Now in order to enter the urine in a state of solution they must necessarily have been in a state of solution in the blood. Inasmuch as they will not remain in solution in an alkaline medium, there is no escape from the conclusion that the blood from which they have been derived is an acid, and a definitely acid, and not an alkaline fluid.

So much being established, we are justified in concluding that the degree of the acidity of the urine is a measure of the degree of the acidity of the blood. This acidity in both cases is due to acid phosphates, salts of H_3PO_4, that is, which like NaH_2PO_4, $CaHPO_4$, and $MgHPO_4$, are still capable of taking up one or more atoms of base. It is therefore obvious that the real, the essential, urinary acidity is phosphoric acidity. Acids of other kinds are doubtless often present in urine, but they are as a rule fortuitous and abnormal, such for example as oxalic acid, uric acid, and other organic acids, which, so far from being desirable constituents, generally indicate disorders of metobolism. The problem which M. Joulie set himself to solve was that of finding a reliable means of estimating this phosphoric acidity in any individual case. Having evolved the process which I shall now describe by observation on the urines of a great number of persons in good health conducted in conjunction with Dr. Nicolaidi, certain normal standards were arrived at, and these standards now serve for comparison as to the sufficiency or excess of the phosphoric acidity in any given urine.

The specific gravity of the urine to be examined is taken at 15° C. If the temperature as registered by the thermometer differs from this figure, a correction is made according to a published table. The total acidity, *i. e.* the acidity from all sources, is now calculated, and is expressed in terms of the amount present per 100 parts of the excess of the urine over that of distilled water at the same temperature. In this way the errors inseparable from the varying amount of water in different specimens of the same person's urine are altogether avoided. The degree of dilution of the specimen, always hitherto a matter of difficulty, may thus be disre-

* 'L'Acidité Urinaire,' par M. H. Joulie.
† 'La Medication Phosphorique,' Octave Doin.

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garded. The excess of the urinary density over that of water is easy to ascertain. Thus, if the specimen in question shows specific gravity 1020, the excess (which is called E) is 20, because the density of water at 15° C. is known to be 1000.

The total acidity is calculated by means of precipitation with a standard solution of sucrate of lime (10 grammes of powdered chalk, 20 grammes of sugar, to 1 litre of distilled water). The figure thus obtained is called A. Thus we have $\frac{A}{E} \times 100 =$ R. A. (*i.e.* the ratio of the acidity; in other words the percentage of total acidity in E).

The normal standard for R. A. as worked out by M. Joulie and his coadjutors is between 4 and 5—as nearly as possible 4·5. But, as we have already seen, this total acidity is from our point of view a matter of secondary importance. What we wish to ascertain is the amount which the acid phosphates contribute to this acidity: in other words how much of this acidity is real, serviceable phosphoric acidity, and how much of it is organic, fortuitous, and undesirable. The next step in the process is therefore to determine the amount of phosphoric acid, combined and uncombined, which is present in our specimen. This is calculated by the classical method,* that with nitrate of uranium and ferrocyanide of potassium and is expressed, as in the case of the total acidity, in terms of the amount present per 100 parts of the excess of the urinary density over that of water at the same temperature. If we call the total phosphatic contents thus obtained P, we have the following formula. $\frac{P}{E} \times 100 =$ R. P. (*i.e.* the ratio of the phosphates; in other words the percentage of total phosphatic contents in E). The normal standard for R P as estimated by M. Joulie and his coadjutors is between 11 and $11\frac{1}{2}$, as nearly as possible 11·17. In order to find what we are in search of, namely the amount which the phosphates contribute to the total acidity, it is necessary to divide R. P. by R. A. thus: $\frac{RP}{RA} = \frac{11{\cdot}17}{4{\cdot}5}$ = 2·45. This represents the normal percentage of active phosphoric acid, of phosphoric acid, that is, which is capable of neutralising an equal atomic weight of base, in E, which is the excess of urinary density over that of water.

It is not necessary to dwell upon the clinical utility of this method of examining the urine. Theoretically, of course, the total acidity may be unduly high or unduly low, and similarly the phosphatic acidity may be excessive or deficient. Practically, however, it has been found from an examination of over 12,000 cases† that the total acidity is seldom on the plus side, and the phosphoric acidity almost never. When the total acidity is excessive and the phosphatic contents are normal or nearly so, excess is due to unoxidised organic acids, and this explanation is borne out by the fact that such readings are invariably associated with gastro-intestinal disturbances, usually in the form of dyspepsia of the sthenic type. When the readings show that both R. A. and R. P. are below the normal, the explanation is simple enough. It is that there is an insufficient quantity of phosphoric acid in the blood and the symptoms are therefore due, in part at any rate, to the deposition of the earthy phosphates in certain organs and tissues, the treatment resolving itself into the exhibition of phosphoric acid in gradually increasing quantities.

The results of an examination by this method are however not always so easy to explain as the foregoing. For instance, the first urine examined for me was as follows:

R. A. instead of being 4·5 was 2·34: R. P. instead of 11·17 was 10·32. $\frac{R.\ P.}{R.\ A.}$ instead of 2·5 was therefore 4·83. There was thus a serious diminution of total acidity, while the phosphatic contents were nearly normal. The significance of this was not at first obvious, but on consideration it became clear that the phosphates, though present in a quantity which was all but sufficient, were present in a form which contributed inadequately to the general acidity; that, in fact, they were present not as acid phosphates (NaH_2PO_4, etc.), but as neutral phosphates (Na_3PO_4, etc.). There was thus no deficiency of H_3PO_4, but merely an excess of alkali, which neutralised the normal surplus of acid; a relative, but not an actual phosphoric subacidity. The indication seemed to be to restore the balance by giving phosphoric acid. This was done with results which up to the present seem to be excellent.

The advantage of this method of examining the urine is that it supplies definite indications for treatment. Some arthritic conditions for example are due to hyperacidity, others to subacidity. With this process to aid us we need no longer rest in any doubt upon such a point. An examination according to Joulie's method will not only establish the fact of hyperacidity or subacidity, as the case may be, but it will give us the degree of the deviation from the normal, and will at the same time unmistakably inform us of the proper line of treatment. It is, so far, in chronic arthritic conditions that it has yeilded the most valuable information, but it is obviously capable of affording information almost equally valuable in various other complaints, especially chronic complaints; for if the urine presents any departure from the normal, one at any rate of the lines of successful treatment will be that which brings about an improvement in this respect.

April 16th, 1906.

* See Bedford, 'Clinical Handbook of Urinary Analysis.'
† Nicolaidi, 'L'Acidité Urinarie.'

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE ON SOME CASES OF ENLARGED LYMPHATIC GLANDS.

Delivered at Guy's Hospital, October 26th, 1905.

By W. HALE WHITE, M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—I have here some patients who have enlarged lymphatic glands, and I thought it might be interesting if, this afternoon, we were to go over some of the causes of chronically enlarged lymphatic glands, but, of course, in doing so we can only consider enlargement which is tangible during life; therefore, it comes to this, that we only can consider the enlargement of glands which are comparatively close under the skin. To-day we shall, therefore, not include deep-seated glands.

Mostly, when lymphatic glands are enlarged, the enlargement is secondary to some disease peripheral to the gland. If you will remember that you will save yourselves many mistakes. The commonest of all is for some septic condition to be the cause of the enlargement of the glands. If you see a man with an enlarged painful gland in his axilla, you may find that he has a suppurating wound in his finger. Another instance of the same point is the enlargement of glands in the groin following gonorrhœa, and the enlargement of glands found in association with a hard chancre. Only the other day I was asked to see a woman, and the medical question raised was as to whether she was strong enough to undergo an operation for extirpation of the breast. There was a doubt as to the presence of a tumour in the breast, but what settled the diagnosis of malignant growth was the detection of hard enlarged glands in the axilla and one above the clavicle. If it had not been for the detection of these glands, the woman might not have had her breast excised. And I saw last week

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a woman who had some malignant disease of her lungs secondary to malignant disease of the breast. The tumour of the right breast was ulcerating and larger than a fœtal head, and there was a mass of glands in the axilla so great that she could not bend the arm less than to a right angle. It was not the doctor's fault that this state of things occurred. She did not tell anyone that she had this malignant disease, not even her husband, because she was so afraid it would have to be cut out, and she had let it go on to the condition which we read about as being common in the days before anæsthetics were used. Another position of enlargement of glands due to growth which you should bear in mind has been called attention to lately, namely, when there is malignant disease in the abdomen there may be an enlarged lymphatic gland over the left clavicle, because that, as you know, is where the thoracic duct enters the vein. There is a beautiful picture published in the 'British Medical Journal' this year by Dr. Stevens, showing malignant disease spreading up the whole of the thoracic duct, and giving rise to an enlarged gland above the left clavicle. And Dr. Raw also publised some cases bearing on this point. It was suggested, when the correspondence was going on about these cases, that sometimes the malignant disease might be disseminated from the abdomen up into the neck by other routes than the thoracic duct. Be that as it may, there is, as many of you have seen, in the wards a very considerable importance attaching to enlargement of the glands above the left clavicle as evidence of malignant disease in the abdomen. Sore throats, again, are a very common cause of secondary enlargement of lymphatic glands. For instance, diphtheria comes to one's mind, and rheumatic sore throat. And in mentioning that I might remind you that the enlargement of glands from rheumatic sore throat is not generally great. Usually it is bilateral. There has been a most interesting case in bed No. 32, Miriam Ward, this month. A woman was admitted with what was thought to be rheumatic sore throat. The glands were only enlarged on the right side, forming a mass behind the jaw. It was so large a mass of glands that we ventured to doubt the suggestion that the enlargement was due to rheumatic sore throat. Finally, the glands broke down, and pus had to be let out, and the pus contained a pure culture of *Streptococcus longus*, showing we were right in believing such a great enlargement of the lymphatic glands could not be due to rheumatic sore throat.

Some of you should feel this little boy's neck, because he shows a very common cause of error among beginners in medicine. Those enlarged glands on the back of his neck are due to the fact that when he came in his head was full of pediculi, and I always remember the case of a patient who came into the out-patients, and the students thought she had Hodgkin's disease until she removed her hat, and then the cause of the supposed Hodgkin's disease was seen. So you must not overlook pediculi as a cause of enlargement of the lymphatic glands. In Miriam Ward at the present time there is a woman who shows another point very prettily, for in her case an enlargement of lymphatic glands is due to leech bites. When she came in she complained of tenderness in the left axilla, and we felt a few enlarged glands. We discovered that the doctor outside had applied some eeches to the side of her chest. The leech bites had become somewhat inflamed, and the enlargement of the glands was secondary to this. In all the cases I have told you of, the enlargement of the lymphatic glands is local, and secondary to disease at the periphery. But this man, who has kindly come down from the ward, illustrates what I want more particularly to talk about this afternoon, namely, generalised enlargement of the lymphatic glands.

Before commencing to tell you about general enlargement of lymphatic glands, it is worth while to remind you that there are a few rare conditions accompanied by enlargement of lymphatic glands which we need not talk about, such as plague and glandular fever, a short specific affection which is over in a few days; nor need we describe what I delivered a lecture about last year, Still's disease, because there the prominence of the joint symptoms is so great that you cannot overlook them. And we need not now think about that rare condition status lymphaticus. It will save our time if we leave all these diseases aside on the present occasion.

The problem now presents itself, what can possibly be the cause of such enormous enlargement of glands as you see in this patient? When we come to describe the case I will mention the particulars to you. In the first place these glands could not be lardaceous; lardaceous disease

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now, thanks to superior surgery and the improved treatment of syphilis, is rare, and there is nothing in this patient that could lead to it; further I have never known or read of lardaceous disease producing such enormous enlargement of the lymphatic glands as you see before you. So we can put that possibility out of court. Whenever you are in doubt about a diagnosis you should think of the possibility of syphilis. I mentioned that the glands in the groin are enlarged secondary to a chancre on the penis, and in the same way the glands in the axilla will enlarge secondarily to chancre on the finger. But in this patient there is not a local, but a general, enlargement of the lymphatic glands, and I do not think syphilis will ever produce such a condition as you see before you. The only general condition of enlargement of lymphatic glands that syphilis will produce is that which you know in the secondary stage of syphilis, in which the glands in the sub-occipital triangle are enlarged, and sometimes the gland above the bend of the elbow. But that enlargement never arrives at the extreme degree which you see in the patient before you. Then there is the possibility that our patient has splenic leucæmia. It is true that in that disease the lymphatic glands do enlarge, but the fatal objection here is that the spleen is not enlarged. So this patient has not got splenic leucæmia. There is another disease in which the lymphatic glands enlarge, namely lymphatic leucæmia. The fatal objection to that being the case is that this patient has not any leucæmia. You will remember that lymphatic leucæmia is a disease in which the lymphatic glands all over the body enlarge, and there is an enormous increase of lymphocytes, so that they form 80 per cent. of the white cells present. But, even supposing we had not the blood to show that there was no leucæmia, we could put this disease aside, for never, I believe, in lymphatic leucæmia, which is rare, do you get such extensive enlargement as you find to be the case here. So that goes to the wall as a possible diagnosis.

Now we come to a disease which is of much more practical importance, namely, can it by any possibility be the case that this patient has tuberculosis of the glands? And when you are thinking of tuberculosis of the lymphatic glands you have to think of two conditions; one where the glands are enlarged at only one part of the body, usually the neck. That is what our forefathers called scrofula. The probable reason why tubercle is so common there is that the tubercle bacillus has an entrance through the tonsil or other parts of the mouth, and these bacilli get deposited in the nearest lymphatic glands. Other instances of the same thing which occur in the body are that the tubercle bacilli from the lung get deposited in the bronchial glands, and the tubercle bacilli from the intestine get deposited in the mesenteric glands. Could this patient be an instance of tubercular deposit in the glands of the neck, dependent on the fact that the tubercle bacilli had got in through the tonsils? I will give you the various points to show that that could not be so. Think of what is going to happen to tubercular glands. Tubercular glands tend to caseate and break down and discharge. If you go into the isolation ward at present you will see a child with a mass of semi-fluid glands on the side of the neck, and there is a discharging sinus. That is what our forefathers called scrofula, and it will lead to a scar. That is the usual course of tuberculosis of the glands of the neck. If you look at this man you will find there is no adherent skin over the glands, there is no softening of the glands, they are not breaking down, they are not caseating. As glands caseate and break down the inflammation causes the glands to become adherent. The glands in that man's neck are not tubercular, because they are discrete. The skin is freely movable over them, it is not red over them, the glands are not soft, and there are no sinuses formed. So we learn that the glands in this patient are not tubercular. There is another kind of tubercular disease of the glands, which is not mentioned at all in some of your text-books, but which you will come across from time to time. It is a condition in which all the glands, or certainly most of the external glands of the body, become enlarged as a result of a deposit of tubercle bacilli in them. It is always accompanied by fever, and is usually fatal within a few months, or at the outside a year. I remember some few years ago a patient Dr. Goodhart and I saw together who presented this condition, and who was, I believe, an instance in point. He had continual fever, he grew thinner, he became weaker, and ultimately died, having enlargement of the glands all over his body. The glands became soft, and one cut into contained pus. Unfortunately

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there was no opportunity of examining it for tubercle bacilli, and a post-mortem examination was not allowed.

Let me read to you also the account of another case illustrating the same disease from the 'Guy's Hospital Reports' of 1873. It was that of a man admitted under Mr. Cooper Foster for disease of the right knee-joint. He had a large mass of glands in the posterior triangle of the neck. He died after amputation of the thigh. At the autopsy the glands in Scarpa's triangle were found to be enlarged, yellow, and putty-like. The inguinal, iliac, and lumbar glands of the right side were all very firm, large, and yellow. Some of the glands in the abdomen were as big as chestnuts, the bronchial glands on both sides were enlarged, they were cheesy, and the disease seemed to be spreading from them into the lung itself. The glands in the right axilla and those on both sides of the neck were in a similar condition. There was miliary tuberculosis of the lungs. Then there follows the record of another case, which was under the care of Dr. Fagge, which I need not trouble you with, or we shall not have time to talk about all we want to get into this lecture. I want you to remember that there is such a disease, because it is easily overlooked, a disease in which the tubercle bacilli affect the lymphatic glands all over the body, making them break down, a general disease accompanied by fever, loss of strength, wasting, and anæmia, and killing within a few months, or at all events at the end of a year. This man before us now cannot be a case of this kind, for in the first place he is not wasting, he has had this enlargement for some time, none of the glands are getting soft, he has not the temperature so characteristic of the disease, and in none of the points I have mentioned does he resemble that disease. But the importance of remembering that there is such a condition I will show you again soon.

Another disease we must consider is this. It was suggested at the bedside that this man might have malignant disease. But he is not ill enough or wasted enough for that. There is a form of generalised sarcoma which tends to affect the lymphatic glands all over the body. Fortunately it is very rare, because it is a terrible disease. The skin becomes adherent over the glands, the glands are very tender, they are liable to have hæmorrhages in them, the patients are very ill, and they soon die. But such a condition is quite exceptional, and this man does not fall into that category. We went over all these points more or less extensively in the ward with regard to him, and finally we came to the conclusion that he had Hodgkin's disease. That renders it necessary that I should tell you something about Hodgkin's disease. We are rather proud of it here, because, as you know, Hodgkin did his work on the subject at Guy's Hospital. He described, in 1832, in the 'Medical and Chirurgical Transactions,'—I have a reprint of them here—six cases from Guy's Hospital of enlargement of the lymphatic glands. I think he was perhaps rather fortunate in having the disease recognised, but, like some other distinguished people, he was fortunate in his biographer. And Hodgkin's biographer, as regards this disease, was Sir Samuel Wilks. What happened was that Wilks, in 1856, not knowing that Hodgkin described these cases, described some himself, and called them leucæmia lymphatica. Then he discovered that Hodgkin had described the condition. Then Wilks described a third set of Guy's cases in 1865, and pointed out that the credit of the first description of such cases belonged to Hodgkin, and the disease has since been known as Hodgkin's disease. And not only was Hodgkin fortunate in having such a biographer, but he was fortunate in that Sir Samuel Wilks was able to point out that, while all Hodgkin's cases were not cases in point, three of them certainly were, and considering that the original paper contains rather a medley of cases, because they are not all examples of the disease, I think Hodgkin was fortunate in getting the wheat sifted from the chaff by Sir Samuel Wilks. There have been many names for this condition. It is most confusing to students. I have a list in some book or another of, I think, nearly a dozen such names. One author says everybody who writes on the subject invents a new name for it. One name which is given to it is lymphosarcoma, a bad name. Another is pseudoleucæmia, which is a still worse name, because there is no leucæmia in the disease. Another name is malignant lymphoma. And there is also anæmia lymphatica. They are well-known names, but let me advise you to discard them all, calling it simply Hodgkin's disease. The advantage is that it is a name which does not commit you to a knowledge of the pathology of

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the disease. As a matter of fact we do not know anything about the pathology of these cases.

Now I will tell you the chief points about the disease. There is no known cause for it. In this patient we detect no cause. It occurs in the first half of life, and is much commoner in men than in women. You see this patient is a young male. He gives no history of any Hodgkin's disease in the family, and that is true of other cases, nor is there any particular family history of tuberculosis. Then what is true of him is true of the disease generally, that until the patients notice the enlargement of the neck they felt pretty well. They will tell you there was nothing much the matter with them until they saw that their necks became enlarged. Another point is that the disease nearly always begins in the neck, and in the case of this man you will see the disease is most prominent in the neck, and the report states that the trouble first manifested itself there. Another thing which this case illustrates, also very well, is that the glands may form huge masses. In this patient there is an enormous enlargement of lymphatic glands. Then there is the fact I have already mentioned, that the glands are always discrete. They never undergo suppuration or caseation, and the skin is not involved unless there is some secondary infection.

We now come to the next point. I have told you the glandular enlargement is most common in the neck, and always begins there, and the glands are always larger there than anywhere else. Other common sites for the glands to be affected are in the axilla. You can all see this large mass in the man's axilla. The glands in the groin are also large enough to be seen at a distance. So this man teaches you the position of the glands which are affected in this disease: largest in the neck, most common in the neck, beginning in the neck. The next point at which the enlargement is best seen is in the axilla, and the enlargment of glands is also seen in the groins. Other positions for the external glands to be affected with Hodgkin's disease are quite rare. Internal glands are often affected, and sometimes they are so affected as to produce all sorts of pressure symptoms, such as diminished entry of air into one lung. Sometimes the affection of them is so great that you can percuss out a dull area in the posterior or anterior mediastinum. Sometimes the affection of glands in the abdomen is so considerable that you can feel them through the abdominal wall, masses of them. But as a rule you do not do so, and you are often surprised, at the autopsy, to find that the glands in the abdomen are much bigger than you thought it possible for them to be. Some of you may remember a woman who was in the ward a little over a year ago, in whom one of the glands in the right groin was so large that she could not flex her thigh, and in consequence the gland had to be cut out.

Soon after the enlargement of the glands has shown itself, these people begin to get weak and somewhat anæmic. This man is somewhat anæmic. I have the blood counts here, the hæmoglobin was 70 per cent. There is usually diminution in the number of red cells, and nothing abnormal in the white cells. It is a simple anæmia, and there is no leucæmia. A point of very great interest is that, if the case is at all severe, there may be some pyrexia. This man is not in the acute stage, and therefore there is no pyrexia. Many of you will remember a girl, whose chart I have here, who was in Number 5, Mary Ward, during the summer. She came in on May 27th, suffering from Hodgkin's disease; at that distance you can see her temperature chart showing an extreme degree of fever. I have never seen a case of Hodgkin's disease with such extreme fever. Her temperature was often up to 104°. Here is the continuation of the chart. The temperature came down for a time and then went up again, as you see. So these charts will remind you that these patients very often have fever, but in this particular case the fever was greater than is usual. Often, too, the spleen is enlarged. In this man it is not. But it is necessary for you to bear in mind that in Hodgkin's disease the enlargement of the spleen is never as great as it is in splenic leucæmia. The liver also may be a little enlarged. The reason for that we shall see directly.

When you come to make the autopsy—because I have told you that the disease is fatal, and usually lasts one to four years—of course you find enlargement of the lymph glands. We are rich in specimens at Guy's, because we have some of Hodgkin's original specimens. Here is one of them. It was taken from a man who was in here under Dr. Bright in 1830, and he forms one of the original cases upon which Hodgkin's paper was based in 1832. Here is another mass of glands

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from one of Dr. Hodgkin's cases in this hospital over seventy years ago. When you examine the glands microscopically you do not find, as some books tell you, a simple hyperplasia of the gland. If the gland has not been affected long you find that the gland is full of a number of lymphocytes, a multiplication of the natural lymph cells of the gland, and that gave rise to the statement that what was the matter with the glands was a simple hypertrophy. But that condition is early, and soon gives way to a chronic condition of the glands which may be said to resemble chronic inflammation, for new fibrous tissue develops in the gland, and it becomes hard. It is a curious but interesting fact, that many observers have confirmed, that these glands contain a large number of eosinophil cells. We do not know why the disease should occur, we do not know whether it is due to a micro-organism. But we know that not only does this enlargement of lymph tissue occur in lymphatic glands, but it also occurs in other parts of the body. Here is the spleen from a woman who was under Dr. Bright in 1828, and it forms one of Hodgkin's original cases. You will see the enlargement of the lymphatic glands at the hilum of the spleen, and, although the specimen has been in spirit seventy years, you will see a number of whitish areas scattered about in the spleen, and those are areas of enlargement of the lymphoid tissue of the spleen. In a recent post-mortem this increase of the lymphatic tissue of the spleen, occurring as you see there in a patchy manner, leads to what is called hard-bake spleen, that is to say it looks as though you have pieces of almond let into the dark sweatmeat, the white corresponding to the new patches of lymph tissue in the spleen. The "hard-bake spleen" is the typical spleen of Hodgkin's disease. Here is a liver from one of the old cases which we treasure in Guy's, and you can see the new tissue of a similar nature, but it is diffused through the whole organ. Therefore, we have now explained the fact that the liver and the spleen may both of them be felt to be enlarged during life. In rare cases most of the lymph tissue in the body is affected; for instance, that of the kidney and of the tonsils, and most strikingly the lymph tissue of the intestine. Here are specimens showing the hypertrophy of lymphoid tissue of the intestine and testicles.

Now, what is the relation of the disease to other diseases? That is a question which has given rise to a great deal of controversy. Some people have tried to prove that in Hodgkin's disease the glands are affected by sarcoma, others that it is a form of tubercle. But I think I have shown you that Hodgkin's disease is a distinct and separate disease. One of the best articles on the subject is by Dr. Dorothy Reed, and is to be found in the 'Johns Hopkins Hospital Reports,' and the conclusions which she comes to are, I think, worth giving you. After examining the published cases she decides that genuine Hogkin's disease has nothing whatever to do with tubercle. She says—"We should limit the term Hodgkin's disease to designate a clinical and pathological entity, the main features of which are a painless and increasing glandular enlargement, usually starting in the cervical region, without the blood changes of leucæmia. The growth presents a specific histological picture, and is not a simple hyperplasia, but there are changes suggestive of a chronic inflammatory process. The microscopical examination is sufficient for diagnosis. Eosinophils are usually present in great number in such growths. The pathological agent is as yet undetermined. Tuberculosis has no direct relation to the subject." And that is the point I wanted to emphasize. I do want to bring home to you this afternoon that Hodgkin's disease is a distinct clinical and pathological entity, having, as far as I know, no relation to other diseases. The next point to direct your attention to is the only complication I need mention, and that has been the cause of a great deal of confusion. A certain number of patients with Hodgkin's disease die of tubercle. They become infected with tubercle towards the end, and as tubercle is found post-mortem, the case is erroneously considered to have been tubercular throughout.

Before leaving the subject, I thought it would be a matter of clinical interest to tell you of one of Hodgkin's own original cases. Let us take No. 4 of Hodgkin's series, and an abbreviated reference to it may well conclude what I have to say about the disease. "Case No. 4 was admitted under Dr. Addison on December 30th, 1829, in the Clinical Ward. He was not at all wasted, but was rather plump than otherwise. He had a pale and peculiar cachectic countenance, which, without minute description, may be suggested to the mind by comparing it to what is seen in some cases of

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confirmed disease of the spleen. The most remarkable feature in his case was the great enlargement in nearly, if not quite, all the absorbent glands within reach of examination, but more especially within the axilla and groins. Those on the side of the neck were scarcely less so. Most of the glands which were within reach were about the size of pigeon's eggs, a few somewhat larger, and others rather smaller. They were of a smooth, rounded, or ovoid figure, and were only moderately firm, rather than indurated. An enlargement was also felt in one epididymis. . . . The glands in the axillæ and neck, as might have been expected, were found (post-mortem) to be prodigiously enlarged, the deepest-seated being in general the largest. . . . These glands were smooth and of whiter colour externally, with a few bloody spots. When cut into their internal structure was seen to be of a light, nearly white, colour, but with a few small interspersed vessels. . . . The glands along the subclavian arteries and about the roots of the bronchi were much enlarged. There were glands on the small curvature of the stomach, several in Glisson's capsule, and a large mass of them along the entire course of the abdominal aorta, and iliac arteries were also greatly enlarged." That is an historically interesting case.

The next thing to draw attention to is the treatment; and that, unfortunately, we can tell you very little about. We know nothing that can certainly influeuce the disease. I usually give the patients arsenic. I have an impression that perhaps the patients are a little better for it, but I am certain it does not avert the fatal issue. You will frequently be asked whether you should excise the glands. It has been most clearly proved to my mind that excising the glands has no influence upon the disease. Excising glands is only justifiable when it is necessary for some mechanical reason. For instance, we were quite right to take away the enlarged gland from the groin of the woman in bed 17 in Mary Ward, because the gland prevented her flexing her thigh. I often tell you about a good-looking young man who got the disease, and the enlargement of the neck distorted him so that he could not go into society, and he became shy of going anywhere. In that case some glands were removed on several occasions. The patient almost demanded it, because he said his disfigurement was so great that life was a burden. But unless you take away the glands for some mechanical or æsthetic reason you should not touch them at all. Therefore, if the patient has no wish for the operation, you cannot urge him. Moreover, you would find on turning back the skin that many more glands were enlarged than you thought. And it requires a surgeon of some presence of mind and intimate knowledge of anatomy to take out a number of enlarged glands from the tissues of the neck. And the last point is that, although as I hope I have made it clear, there are several perfectly distinct diseases of lymphatic glands, you will come across cases which it is very difficult to place in one category or another. But they are rare. It may be that such cases are examples of disease not yet known to us. The most common are those which it is difficult to decide whether they should be called splenic leucocythæmia, or whether they should be called Hodgkin's disease. Here is a case in point. The patient was in the hospital in May 1890, a man, æt. 25 years, who had on admission an enlarged gland above Poupart's ligament. The spleen was down to the umbilicus, the liver one inch below the ribs. The blood showed white corpuscles one to every fifty red. He frequently had a high temperature, usually 101° or 102°. He improved, and went out. He was re-admitted on March 9th, 1891, and then the spleen and liver were about the same as before. There was still an increase of white cells, but not so great, the lymphatic glands in the neck were enlarged, and he sank and died. I made the post-mortem, and found this condition of affairs. The retro-peritoneal glands were enormously enlarged, one in the pelvis above Poupart's ligament was two inches in diameter. It showed some hæmorrhages in its interior which had formed cysts. There were several spots of caseation. The glands in the neck were considerably enlarged on both sides. The spleen weighed forty-six ounces and contained four or five infarcts. It and the liver presented a typical appearance of Hodgkin's disease, and that was in addition to the infarcts. The kidneys were very pale and contained infarcts. The tonsils were normal. There was an increase of lymphoid tissue in the medulla of the bones, and there were masses of lymphoid tissue in the lungs. There was lardaceous disease of kidneys and liver. That was an instance in which the enlargement of the glands strongly suggested that the patient had Hodgkin's disease. The liver and spleen and general increase of lymph tissue looked like Hodgkin's disease. On the other hand there was such a considerable excess of white cells that it was a difficult case to classify, a difficulty increased by the caseation of the glands and the lardaceous disease.

April 23rd, 1906.

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WITH MR. F. C. WALLIS AT CHARING CROSS HOSPITAL.

GROWTHS IN THE RECTUM.

GENTLEMEN,—The various malignant growths that occur in the rectum were mentioned by me last week, and this afternoon I will discuss the diagnosis and treatment of these cases.

Malignant disease of the rectum occurs at almost any age. I have seen it well marked at the age of fifteen. In these early cases there is more often than not a history of heredity, and the symptoms which the patient complains of are pain, tenesmus, and loss of blood, and some symptoms in a well-marked case depend upon the situation of the growth. If it is quite low down, the pain, feeling of weight, and difficulty with the bowels are definite, and occur earlier than when the growth is higher up. If the anus itself is involved the pain occurs sooner and is much more acute than in cases where the growth is higher up. In these cases the inguinal glands are early involved, and whether obviously so to the touch or not, they should in all cases be removed. When the growth is higher up the bowel the lack of any early symptom is one of the unfortunate points in these cases, and neither pain, discomfort, nor hæmorrhage are, as a rule, sufficient for the patient to seek advice, and it is not until persistent diarrhœa occurs (apparently causeless), with loss of flesh, that advice is sought.

FIG. 1.

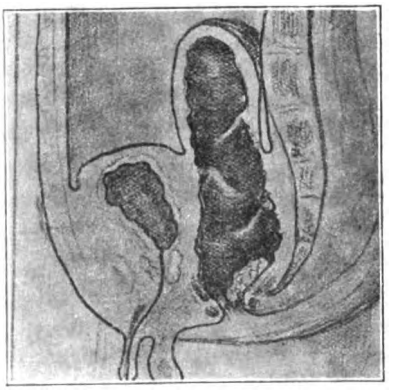
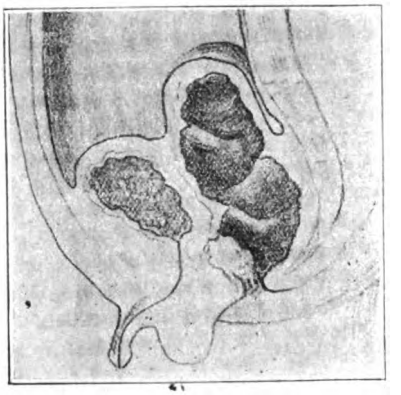


FIG. 2.



difficulty with regard to the action of the bowels. These symptoms are also the common signs of hæmorrhoids, and unfortunately, it is still too frequently the custom to treat these symptoms without making any proper examination.

It is only fair to say, however, that medical men recognise much more now than even a few years ago how absolutely imperative it is not to make any diagnosis nor to suggest any treatment without first making a proper rectal examination in these cases. This should be a golden rule, which no feelings of mock modesty on the part of the patient or fear of wounding the susceptibilities of the patient on the part of the medical man should prevent being carried out.

Malignant disease is much more common after forty-five, and in a well-marked case there should not be any difficulty in making a diagnosis. The

Recently I operated on a patient who on account of former troubles was rather on the look-out for anything wrong with himself, and because of a certain amount of bowel discomfort and looseness consulted his medical man, who examined the rectum, and found a large excavated ulcer, with raised edges, about the size of a florin.

Now, the interesting point is that fifteen months before I had seen this man in consultation concerning some pruritis ani, and then there was no suggestion of any ulcer or growth whatever. In this instance a very early case was caught, and if the microscope speaks truly a good result may be permanent; but such good fortune is rare.

When a patient of middle age and over complains of some indefinite trouble with his lower bowel,

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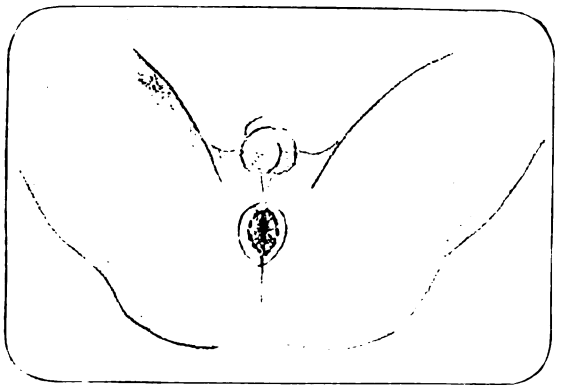
and especially if diarrhœa occurs in an otherwise healthy individual, it is always suggestive of a malignant growth, and a thorough examination should always be made.

FIG. 3.



A raised, hard edge, with an ulcerated area beyond it, partly or entirely encircling the bowel, is what one always finds in such cases.

There are two points which I wish to emphasize:

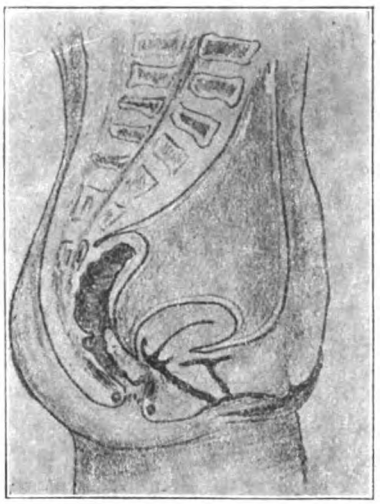
(1) That no diagnosis as to what can or cannot be done should be made in an apparently advanced case until the patient is examined under an anæsthetic, when a growth which apparently was quite fixed will often be found to be freely movable.

(2) In women do not mistake the cervix uteri felt through the anterior rectal wall for a carcinoma; this has not infrequently been done.

The treatment of carcinoma recti depends upon—(*a*) the situation of the growth, (*b*) the mobility or fixation of the growth, (*c*) the general condition of the patient.

(*a*) *The situation of the growth.*—When the growth is situated in the anal portion of the bowel it is rarely seen before the surrounding muscles are involved. Growths situated in the anterior segment may involve the prostate in the male. Figs. 1 and 2 show these growths.

FIG. 4.



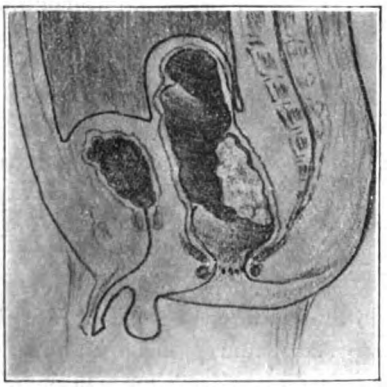


FIG. 5.—Infra-peritoneal.

FIG. 6.—Peritoneal.

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Fig. 3 shows the incisions to make when operating in this situation. The outer line is the one to use when there is no chance of saving the sphincters. The inner line (dotted) is the incision to make when the growth can be removed *per anum*, and

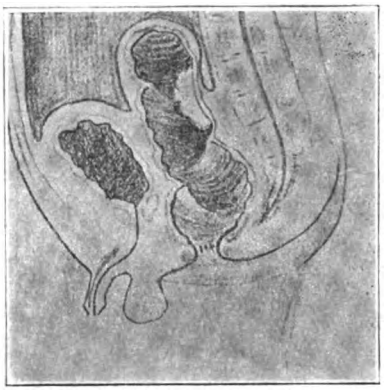
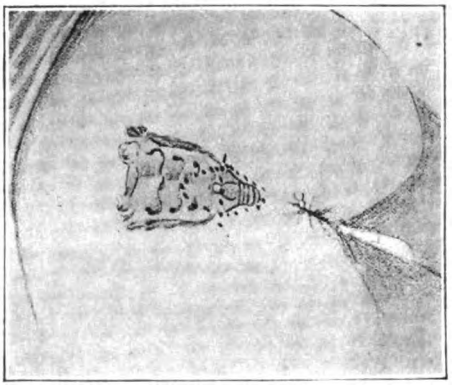


Fig. 7.—Sigmoidal.

when the sphincters are not involved. It is better to dissect off the whole circumference of the bowel, even when the growth is a limited plaque. The possibility of complete removal is much more likely, and it is easier to get above the growth by this method.

Growths situated as in Fig. 4 are best removed

Fig. 8.



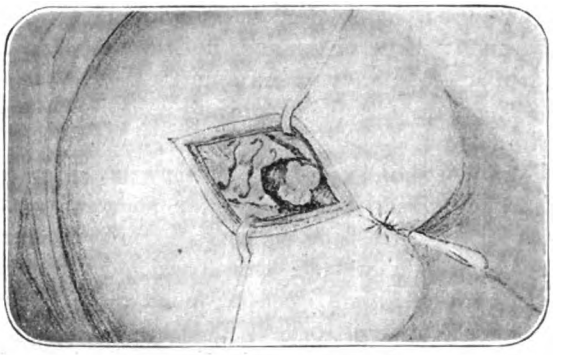
by dividing the posterior vaginal wall; any part of the vaginal wall or cervix uteri affected can be removed at the same time. Douglas's pouch can be opened, and the upper limit of the growth easily arrived at.

Figs. 5, 6, and 7 show the various situations where the growths may occur.

The infra-peritoneal and peritoneal forms are best operated upon by the coccygeal or sacral method. Fig. 8 shows the sacrum with the dotted lines for either the removal of the coccyx, or an elliptical portion o the sacrum as well. The removal of the portion of the sacrum shown in the dotted line gives a large amount of extra room without the destruction of the tissues necessary if a trans-sacral section is made. The possibilities of this are shown in Fig. 9.

When a growth which can be removed partially or entirely encircles the bowel, the ideal operation is to free the growth through the sacro-coccygeal opening, without opening the bowel, and to separate the bowel sufficiently to be able to bring healthy mucous membrane above the growth down to the anus. The patient is now placed in the lithotomy

Fig. 9.



position and the mucous membrane is removed by an incision around the anus, as shown in the dotted line of Fig. 3. This dissection is continued up until the freed bowel in the sacro-coccygeal opening is arrived at. The bowel containing the growth is now pulled down outside the anus, and removed by cutting through healthy tissue above the growth. This healthy cut edge is sutured to the anus. The sacro-coccygeal opening is lightly packed with gauze, and the incision partially closed by interrupting sutures.

It may be, and often is, undesirable or impossible to do all this at one operation, in which case a temporary sacral anus is made, and later on the operation is completed as shown in Figs. 10, 11, and 12.

To remove a growth by the combined abdominal and anal method, the growth must be in the sigmoid

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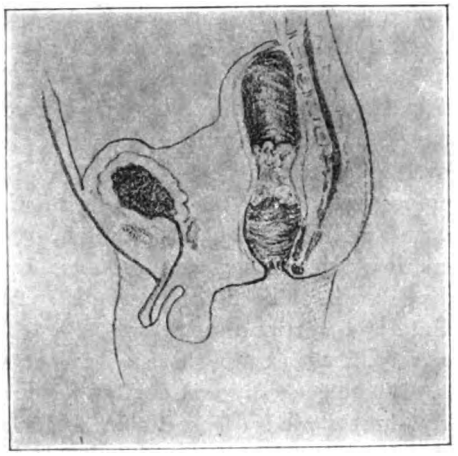
part of the bowel and freely movable, the pelvis must be sufficiently large, the patient not too fat, and preferably a female. Under these conditions excellent results may be obtained from this operation. But it will only be available for a small proportion of these cases, and, as a general rule, the sacral-anal method described above will be found the best and least dangerous.

FIG. 10.



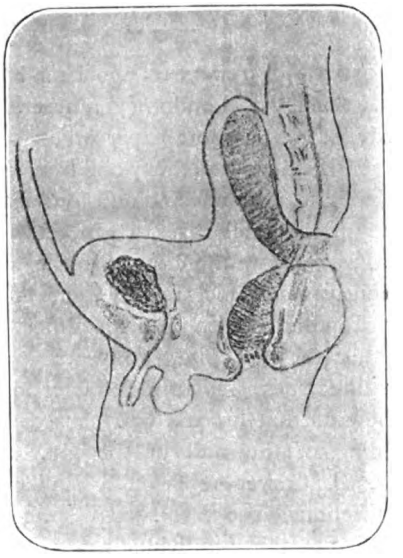
(*b*) When, after complete anæsthesia has been obtained, the growth is found firmly adherent to the surrounding tissues, no attempt at removal should be made. The treatment then depends upon :

(*c*) *The general condition of the patient.*—Even in apparently favourable cases for removal the patient's general recuperative powers are often much impaired, and the resisting powers of the tissues are so much diminished that in spite of every care, and even when the peritoneum has not been opened, peritonitis occurs and rapidly kills the patient. If these cases are examined post mortem it will be found that the disease was not limited to the local growth, but had already involved the lumbar glands, and secondary growths will probably be present in the liver.

In all such cases the general vitality is much lowered, and the great resisting power of the peritoneum, upon which one relies so much in operating in these cases, fails altogether with fatal results.

When the growth is fixed, if the patient is not suffering much pain and the bladder is not involved, if there is no hæmorrhage and no obstruction, colotomy should be delayed as long as possible

FIG. 11.



But if there *is* much pain and the patient's temperature and general condition indicate that septic absorption is taking place, colotomy should be

FIG. 12.

forthwith resorted to, and as soon after as possible the lower part of the bowel should be irrigated.

April 23rd, 1906.

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ON CHRONIC COLITIS IN CHILDREN.*

By EDMUND CAUTLEY, M.D.Cantab., F.R.C.P.Lond.,
Physician to the Belgrave Hospital for Children and the Metropolitan Hospital.

GENTLEMEN,—A few years ago I read before the Medical Society of London ('Transactions,' 1901, vol. xxiv, p. 206) a short paper on an epidemic of acute colitis. Six out of thirty children, living in the same building, were affected and four of them died. In one of the fatal cases, a girl, æt. 5 years, the large intestine from the cæcum to the anus was so enormously thickened, by an acute proliferative catarrhal process as to form an almost rigid tube. A brother of this child, two years younger, died after a like attack, similar in character and duration, but the catarrhal process had caused no definite thickening of the wall of the gut. Last year Dr. A. E. Carver reported a similar epidemic ('Transactions,' 1905, vol. xxviii, p. 237). From twenty-five to thirty individuals were attacked, ten of them severely. Seven of these ten were under ten years of age, all girls except one, and of the seven no less than six died.

Chronic colitis does not appear to have been discussed before this Society, at any rate recently, for I can find no record of it in the 'Transactions' during the sixteen years of my membership, except one clinical case. This was a girl, æt. 4 years, whom I brought to a clinical meeting as possibly affected with chronic follicular colitis ('Transactions,' 1901, vol. xxiv, p. 272). For a year she had been ill. During six months the stools had been frequent, loose, and very offensive, and for two weeks they had contained small black particles of clotted blood. Vomiting occurred daily, but there was practically no abdominal pain or distension, and no fever. At the autopsy one month later two tuberculous ulcers, causing stricture, were found in the colon; extensive ulceration of the cæcum; multiple ulcers in the small intestine, with a little adhesive peritonitis at their bases, but no caseous mesenteric glands. A small, old caseous nodule was found at the apex of the left lung. Tuberculous ulceration of the colon is usually part of a general alimentary tuberculosis. I do not propose to refer to it further and have merely mentioned the case as an illustration of a diagnostic difficulty. Dysenteric ulceration has never come under my notice in children in this country.

The rarity of chronic colitis in children is evident from a recent paper by Hale White, based on sixty cases from hospital and private practice. Of these sixty cases, which he names "membranous colitis," only one was under twenty years of age, a child, æt. 4 years, with catarrhal colitis. She had passed huge casts for two years and had not improved under treatment.

I propose to consider the disease in relation to its several varieties, although they shade off more or less into each other, and cannot always be strictly defined. The nomenclature is somewhat indefinite and worthy of criticism. Probably all but a very few cases begin as acute catarrhal colitis. These generally end fatally or recover in a fairly short period of time. A few become chronic, ending in chronic catarrh, with or without ulceration, or in membranous colitis. The catarrhal form has received other names, such as mucous, muco-membranous, or membranous colitis.

Chronic catarrhal colitis is characterised by the presence of mucus in the stools. The mucus is discharged in glairy masses, which are often described as resembling slime, jelly, or white of egg. The masses of mucus may be so coherent as to float out in water and look like membrane. They differ from membrane in their appearance and in readily disintegrating on stirring. Microscopically, the masses are composed of pure mucus, with denser portions containing desquamated epithelial cells, leucocytes, blood-cells, triple phosphates, and granular and fæcal *débris*. To call this affection a membranous colitis is likely to lead to confusion. Sometimes the symptoms are so slight that the affection is overlooked. Generally the diagnosis depends on the evidence derived from the examination of the stools. Blood may be entirely absent. Usually a few red cells are found microscopically.

The disease may follow an acute attack. Thus, a girl, æt. 2 years, had had acute ileo-colitis two months before I saw her. The attack was severe. It had lasted for ten days, with fever up to 104° F. She was still passing unduly frequent stools con-

* Delivered before the Medical Society of London, Chandos Street.

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taining mucus and occasionally a little blood. In two weeks she quite recovered.

Sometimes no cause can be ascertained. Last November I saw a little girl, æt. 4 years, living a healthy, well-regulated life. For a year she had been passing mucus in the stools, on and off, being worse during the last six months. Apparently she was a healthy, well-nourished child, not at all anæmic. The stools were sometimes quite characteristic, macroscopica ly and microscopically. Occasionally a stool would contain a wineglassful of almost pure mucus. No cause could be ascertained. She had had no acute attack, had not suffered from constipation, and had not been exposed to chills. When seen a few days ago she had quite recovered. No mucus had been seen for over a month.

Ulcerative colitis.—Catarrhal colitis may end in ulceration. Of this there are two types, the catarrhal and the follicular ulcer. The latter is a small, punched-out u cer, due to breaking down of the solitary follicles. Usually follicular ulcers are small, multiple, and are found in infancy in those cases of acute colitis which run on for more than three weeks. Sometimes several unite to form larger ulcers, but the large ulcers are more commonly of the catarrhal type. A certain amount of superficial abrasion or ulceration is seen in the acute catarrhal type, and it may extend to form an extensive, fairly superficial ulcer, or more than one ulcer may be present.

Ulcerative colitis is characterised by loose, offensive stools, from two to four or more daily, and the passage of moderately bright blood, and perhaps mucus. Pain and tenesmus may be present. In a few cases tenderness is found, or a feeling of soreness is present, at various parts of the colon.

The following case is illustrative of this variety and its probable origin in an acute attack of ileo-colitis. A boy, æt. 9 years, was seen by me in consultation with Dr. J. Smith, of Putney, last October. Eight months previously he had had an attack of diarrhœa and vomiting. This was followed, four months later, by another and worse attack of the same nature, and he had never been well since. The chief symptoms were slight febrile attacks, with pain and vomiting, followed by the more frequent passage of loose, rather offensive stools, containing mucus and brightish-red blood. Between the attacks the stools were somewhat the same, but less frequent. The general condition was pretty good. He was rather pale. Nothing abnormal was found on physical examination except a little tenderness along the transverse and descending colon. We saw him again, in conjunction with Dr. Sidney Martin, one month later. There was no improvement. Injections of nitrate of silver were prescribed, and since then he has definitely improved. He digests his food better. The stools are less frequent and contain less blood.

A second case in a boy of the same age is interesting, but the diagnosis is less certain. I saw the boy in consultation with Dr. Graham Morris, of Wallington, in January, 1902. He had had diarrhœa for a month, with fever up to 103° F., septic in type. The stools contained much mucus and some brightish blood. One month later the temperature was keeping at a lower level, the stools were less frequent, and contained only a little blood. Under an anæsthetic a few enlarged mesenteric glands could be felt. After a long illness the boy got well. I saw him in October last, looking perfectly healthy and strong. In both these cases, and especially in the second one, there may be a little doubt as to the exact form of ulceration present. The possibility of tuberculosis cannot be absolutely excluded.

Membranous colitis.—The membranous type is secondary to an acute attack or may possibly be primary, due to the pneumococcus or the various species of septic organisms. No doubt a diphtheritic infection might occur, but I have never seen diphtheritic membrane extending beyond the pylorus, and infection *per rectum* is very unlikely. I regard the process as simply a more severe form of catarrhal colitis. The primary affection may be a simple catarrh, and the membrane may not form until it has existed for some months. The following case is a striking instance of the course and development of the disease. No doubt this form of colitis is extremely rare. Like most other so-called rare diseases, it will probably become much more frequent when once attention is attracted to it. The membrane is not disintegrated by stirring in water, as that of catarrhal colitis, and its presence may be associated with the passage of mucus and even a little blood.

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The case is that of a boy, who was born on June 7th, 1903, and weighed 9½ lbs. At first he was fed on sterilised milk, cream, water, and sugar. He did not digest this properly, was constipated, and began wasting. At twelve weeks he was put on a dilute mixture of Allenbury No. 1 food, and was fed on this for three or four weeks, although the stools became loose and unsatisfactory. Peptonised milk was tried for one day without success. His diet had consisted since then of the Aylesbury No. 1 humanised milk, and of this he was taking 4⅓ ozs. every two and a half hours when he was brought to see me on October 24th, 1903. Since the age of three months the condition of the bowels had been persistently unsatisfactory, and he had passed mucus. For one week the stools had varied in colour, were large, solid, and contained much curd and mucus. Except flatulence and regurgitation, there had been no other symptoms. The previous treatment had consisted of castor oil, bismuth, and rectal irrigations with solutions of boric acid.

On examination nothing abnormal was found, save a moderate degree of flabbiness and malnutrition. He weighed 10 lb. 10 oz. The diagnosis was catarrhal colitis, and attempts were made to find a suitable diet for the child. A mixture of cream, peptonised milk, water, and lactose, four ounces being given every three hours, made matters much worse. The stools increased in frequency, up to seven to ten in the twenty-four hours, and were generally of the same character, though occasionally a normal stool was passed. The appetite was good regurgitation and flatulence were less.

On account of the diarrhœa he was given alternate feeds of albumen-water and rice-water on October 31st for three days, and got much better. A diet of cream and whey was then tried, but the stools became bad again. On November 11th condensed milk, "Ideal" brand, was tried, with the result that an attack of acute diarrhœa, with rapid pulse, sunken eyes, collapse, and rapid loss of weight, came on and lasted for two days. This attack caused a loss of weight of 14 oz. in three days, and was nearly fatal.

During recovery he was fed on Allenbury No. 1 food, albumen-water, and a little bovril. The food still proved unsuitable and so did whey, given in two-ounce feeds every two hours. Malted milk and Allenbury food were also unsatisfactory, and the outlook was distinctly gloomy.

On November 26th he weighed an ounce under 9 lb. He was now fed every two hours on two-ounce feeds of albumen-water and two-ounce feeds of Horlick's malted milk, one feed of the former for every two of the latter. Small frequent doses of castor oil were given.

From this time onward he began to improve, though his progress was interrupted at times by relapses of varying severity. On December 6th he had a slight relapse for a few days, necessitating a reduction in the diet. He had gained 8 oz. in weight. On December 9th he was better, and was ordered a mixture of creosote, yolk of egg, glycerine, and olive oil. This mixture he still takes. On December 20th he weighed the same. His stools were less frequent and did not always contain mucus. His diet had been gradually increased up to four-ounce feeds every two hours.

During the next month his progress was slow but satisfactory. He cut two teeth. The stools numbered two to four daily and were often free from mucus. On January 31st, 1904, he weighed 10 lb. 14 oz. On February 19th he first passed membrane, a tubular cast about 4 inches in length. Thin shreds and pieces of membrane were found in the stools for the next few days. Fretfulness, flatulence, regurgitation, and vomiting were noted. On February 24th diarrhœa reappeared, without mucus or membrane in the stools for a few days. On February 28th membrane and a few specks of black blood were present in the stools. The child weighed 11 lb. 14 oz., but now lost weight for a few days. Gradually the stools became less frequent. On March 27th he had gained 18 oz., and had cut more teeth. On April 24th he had gained 21 oz. The addition of small quantities of cream and milk to some of his feeds had shown that he could not yet digest milk. On May 29th he had gained 28 oz. and had eight teeth. His diet consisted entirely of malted milk. On June 26th he had gained 15 oz. Small quantities of potatoes, gravy, and biscuits were given. On July 31st he had gained 13 oz. The diet was further increased by the addition of rusks and milk puddings. He had ten teeth. On August 28th he had gained 13 oz. and cut two more teeth. On September 28th he had gained 10 oz. He had fifteen teeth. A large quantity of membrane was passed after a dose of

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castor oil. On December 21st he had gained 28 oz. Three days later he became irritable, passed more membrane, and lost weight rapidly for a few days. His subsequent progress was satisfactory. On March 12th, 1905, he weighed 24 lb. 7 oz. In April he had an attack of follicular tonsillitis. In July it was noted that no membrane had been seen for a month. At present he is a strong, healthy, well-grown boy, and does not look as if he had ever had a day's illness. Occasionally shreds of fine membrane are found in the stools.

This case is remarkable in many ways. It is evident that the primary condition was one of catarrhal colitis or ileo-colitis. The presence of membrane was not suspected until its appearance, seven months after the mucus was first noticed in the stools. It was associated with the passage of mucus in varying quantities, and at one time with the passage of small specks of blood, which looked as if they might have come from follicular ulcers. For a year the lower bowel continued to form thick membrane. After a dose of castor oil a considerable quantity of thick membrane would be evacuated, and then little or none would be seen for some weeks. During this interval it formed again, and was again cast off. The indications for giving the oil were fretfulness and irritability of the patient, discomfort, and perhaps regurgitation or vomiting. The pulse-rate also generally increased in frequency, and during the early stages of the illness formed a fairly accurate guide to the severity of the condition. For some weeks it remained at 120 per minute or more. A fall to below 100 always indicated improvement.

The diet of albumen-water and malted milk proved the only one suitable in this particular case, but I am not prepared to recommend it for every patient similarly affected. Although it was continued for six months, and during the last month of this time was limited to malted milk alone, the child did not develop scurvy, rickets, or anæmia. Teething also was unaffected. I attribute this result to the antiscorbutic properties of albumen-water and yolk of egg, to the antirachitic substances in the egg and the olive oil, and the iron-containing proteids in the egg. At one time there was a little swelling and discoloration of the gums, which I was not certain about. It might have been scorbutic or merely due to teething. This was in the middle of March, 1904. Some orange-juice was then added to the diet.

The duration of the case is extraordinary. As a rule these patients are more likely to die than get well. The disease must be very rare, for it is the first really chronic case which has come under my notice. The form of colitis usually called membranous is the catarrhal variety, and this generally proves fatal in acute attacks or persists for a time as a mild catarrh.

The drug treatment consisted in giving castor oil and grey powder to act on the bowels and liver; bismuth in large and frequent doses to control diarrhœa; and the creosote mixture to which I have already referred, as a food and an intestinal antiseptic, probably its chief value was nutritive.

The successful result must be attributed to the careful dieting and nursing. A hospital case would probably have died. In this instance the mother devoted herself to the child, was absolutely trustworthy in carrying out directions, and peculiarly observant of slight changes in the character of the stools and the effects of alterations in the diet.

Diagnosis.—The different varieties must be distinguished by the characters of the stools. If only mucus is present, the affection is catarrhal. The presence of a slight amount of blood under the microscope does not necessarily indicate ulceration, but if there is enough to render it visible to the naked eye ulceration is present. Follicular ulceration is probable in children under two years of age. After that ulceration is more likely to be catarrhal. The differences between mucous casts and membranous ones are readily appreciated when once they are seen.

In all cases inquire for a past history indicative of an acute attack and examine the child thoroughly for evidence of tuberculosis. A rectal examination should never be omitted, as mucoid and bloody discharges sometin. · result from rectal affections.

The locality of ulceration is difficult to ascertain, but a certain amount of weight can be attached to soreness or tenderness in the different regions of the colon. Probably it is most frequent in the descending and least frequent in the ascending colon.

Prognosis.—I am inclined to the view that the prognosis is good in the catarrhal cases and not unfavourable in the ulcerative and membranous forms, but in these the illness is sure to be a very prolonged one. Unfortunately, I have no post-mortem

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evidence to bring forward, and this fact in itself indicates either great rarity or small fatality, possibly both. Ulceration is not deep enough to cause perforation, and probably does not give to stricture. These are two possible sequelæ. In the membranous type chronic and eventually fatal marasmus may ensue.

Treatment.—The treatment of these cases must be considered under the heads of diet, general management, drugs, and local applications. As a rule milk, predigested if necessary, is the best diet, but occasionally it proves absolutely unsuitable. The object to be aimed at is to give a nutritious diet which contains as little waste matter as possible.

Infants should be fed on whey, albumen-water, rice-water, condensed milk, malted milk, peptonised milk, or mixtures of the above. The number of feeds and the amount of each must be rigidly regulated, and careful observation kept on the general state of the child and the characters of the stools Too much or unsuitable food may be indicated by fretfulness and irritability before there is definite change in the motions.

Older children should be fed in the same way or on undiluted milk, if they can digest it, and in addition can be given raw or lightly cooked eggs, Benger's food, malted foods, jellies, sugars, pounded meat or fish, and mashed potato. The more frequent the stools and the greater the quantity of mucus, blood, or membrane in them, the more carefully must the patient be dieted. Gradually the diet is increased, according to the progress of the case, careful watch being kept on possible ill effects. Generally speaking, the patient should be kept in bed, but this is a matter which can be only decided in each special case. Trained nurses are an immense advantage. Otherwise it is almost impossible to get the directions as to diet, fresh air, etc., efficiently accurately carried out. Weeks of careful treatment may be nullified by the injudicious administration of some unsuitable food.

In the mild catarrhal cases there is no objection to an open-air life in a warm, dry climate. Clothing should be warm, the abdomen specially protected, and care taken to avoid chills and damp feet. Keep the bowels open. Small doses of calomel or grey powder for a week at a time seem beneficial, as antiseptic and antifermentative. Oleum ricini, Apenta water, or sulphate of magnesia or sodium, should be given in the early morning before breakfast. On the whole I prefer the daily saline aperient in most cases.

Of *drugs* for internal medication it is difficult to speak confidently. I am particularly fond of a mixture of yolk of one egg, an ounce of glycerine, and one or two ounces of olive oil, with ten to fifteen minims of creosote. Of this one or two teaspoonfuls may be given three times a day for a long period. It is most useful in infants. The creosote prevents the mixture going bad, and may have some beneficial influence on the alimentary tract. If the bowels are at all loose or if blood is passed, large doses of bismuth should be given every two to four hours. Of other internal remedies I can only suggest the possible value of petroleum as a lubricant and protective in cases of ulceration. The resources of pharmacy may be able to provide an antiseptic compound of petroleum which is not absorbable.

It is of great importance to remember that drugs given by mouth have to go a long journey before reaching the site of the disease, and that the benefits derived from them may be more than counterbalanced by digestive disturbances to which they may give rise.

Local treatment.—Irrigation of the colon is rarely advisable except in the ulcerative cases. It must then be tried, if simpler measures fail. Irrigation with a gallon of salt solution, one drachm to the pint, or a similar solution of boric acid, can be tried in all cases at bedtime, with the object of emptying the lower bowel and giving it a lengthened period of rest. In prolonged catarrhal cases an astringent such as tannic acid can be used. In the membranous form I should not expect any benefit from irrigation.

For prolonged ulceration silver nitrate injections, gr. $\frac{1}{4}$ to 1 to the ounce, according to the age of the child, may be tried; from a pint to a pint and a half should be injected. The colon should be washed out previously with saline solution.

Of further measures I have no experience. In adults a few cases have been treated by opening the cæcum or appendix, and irrigating daily from above downwards. The cæcal opening is unsatisfactory because of the presence of an artificial anus in the right groin, the difficulty in closing it, and the irritation and partial digestion of the skin by the intestinal contents. The appendix has none of these objections. It can be clamped, and fluid can be injected through it daily. Subsequently it is excised. Such a line of treatment may be necessary in ulceration of the ascending colon and may prove successful when all other measures have failed. I merely mention it as a kind of last resort in apparently hopeless cases.

April 23rd, 1906.

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THE SYMPTOMS AND SURGICAL TREATMENT OF GALL-STONES.*

By FREDERIC EVE, F.R.C.S.

IN a single lecture it is only possible to touch on the principal features of cholelithiasis. So much work has been done on this subject, during the last decade especially, that we appear almost to have reached finality as regards symptomatology and treatment. Yet it may be useful and not uninteresting to busy practitioners to place before them a *résumé* of the subject which may serve at any rate the purpose of "marking time."

Calculi may exist in any part of the biliary apparatus, including the hepatic duct within the liver itself, but are most commonly formed in the gall-bladder.

Symptoms of stone within the gall-bladder.—It is well known that stones may remain in the gall-bladder for years and produce no symptoms. Or the symptoms are very slight, consisting of dyspepsia, so-called "spasms," with a feeling of discomfort, weight, and indefinite pain in the right hypochondrium.

In typical cases there are repeated attacks of hepatic colic. Probably in the majority of cases calculi in the gall-bladder produce no symptoms until septic infection has taken place by way of the intestine. Kehr writes: "The idea that a calculus does not cause pain until it has left the gall-bladder is a mistake. Most attacks of colic are caused by calculi or inflammatory processes within the gall-bladder itself." In the following case the symptoms closely resembled those of calculus in the common duct, although the gall-bladder contained stones, and the cystic duct was closed. A woman, æt. 53 years, had suffered with severe abdominal pain for one week and jaundice for four days. The

* Delivered before the Stratford Branch of the British Medical Association.

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pain was very acute, doubling her up and causing vomiting. At the operation the gall-bladder was much shrunken, but no stone could be felt. The common duct was opened and a probe passed freely into the hepatic duct and intestine, but no stone was found. The gall-bladder was then opened and three small stones were removed. The cystic duct was occluded. It was thought advisable to drain the biliary apparatus by a tube placed in the common duct. The patient made a good recovery. The presence of jaundice in this case may be explained by catarrh of the common duct set up by the irritation within the gall-bladder. Patients whose gall-bladder contains large numbers of small calculi often suffer the most pain and inconvenience. The stones are usually about the size of a buck-shot and are continually passing along the common duct, producing the characteristic symptoms. I have removed as many as two hundred small calculi from the gall-bladder.

Stones impacted in the cystic duct produce well-marked symptoms of hepatic colic. This fact was manifested in a woman, æt. 35 years, whose gall-bladder I removed for calculi impacted in the cystic duct, the gall-bladder itself being empty. For seven months she had suffered with attacks of well-marked colic, four having occurred during the six weeks preceding the operation. Jaundice followed one attack. On opening the gall-bladder several stones were found firmly impacted in the cystic duct, but there were none in the bladder itself. I could not dislodge the stones and therefore performed cholecystectomy.

It will be convenient to discuss in detail the symptoms produced by stone in the gall-bladder in relation with the various conditions of the gall-bladder met with in cholelithiasis.

The conditions of gall-bladder met with are :

Dropsy.—The bladder is distended with mucus, not bile, for the cause of the condition is usually impaction of a stone in the cystic duct. Symptoms are sometimes very slight, or entirely absent, the gall-bladder not having become infected with bacteria, or if infected the organisms are of low virulence. In fact, the accidental discovery of a tumour in the abomen often first brings the affection to light. This was the case in the patient from whom this specimen was removed. The gall-bladder, which was successfully removed, was five inches in length and reached considerably below the umbilicus. The patient, a very neurotic woman æt. 39 years, had suffered with indigestion, and pain chiefly across the abdomen below the umbilicus. A stone the size of a filbert was impacted in the cystic duct.

Empyema and cholecystitis, in other words suppuration of the gall-bladder due to infection. — The symptoms associated with this condition vary as much as the symptoms produced by appendicitis. The less acute cases are usually described as suppurative cholecystitis or empyema of the gall-bladder, the more acute cases as phlegmonous cholecystitis. The following case, that of a woman æt. 47 years, illustrates all the symptoms associated with suppurative cholecystitis. She was suddenly attacked with pain in the right hypochondrium and vomiting. The vomiting recurred next day. She complained of pain, extending from the right hypochondrium, around the lower part of the chest, to the angle of the right scapula. On admission the abdomen was somewhat distended, the upper part of the right rectus was rigid, and there was extreme tenderness just below the costal margin, where an indefinite swelling, moving with respiration, was felt. Pulse 96, temperature ranging from 100° to 102° F. Cholecystotomy was performed ; the gall-bladder contained pus and 94 stones. A culture of the pus showed *Bacillus coli*, and a few streptococci. The diagnosis in such a case is obvious, the chief points being, the character of the pain, its sudden onset, with tenderness below the tip of the ninth costal cartilage. This is the so-called "biliary point," which is quite as distinctive of cholecystitis as McBurney's point is of appendicitis. In this case also the presence of a distended gall-bladder clinched the diagnosis. A swelling below, and continuous with the liver, in these cases is often chiefly made up of thickened omentum adherent to the gall-bladder, and may feel quite hard and indurated, although the omentum surrounds a gall-bladder which is filled with pus. The position of the swelling also varies somewhat. Normally a distended gall-bladder extends obliquely downwards and to the left, towards the umbilicus. In a case recently operated on, however, I found an indurated mass extending vertically downwards from the liver, considerably external to the linea semilunaris. I thought it probably a Riedel's lobe, but it proved to be a dilated gall-bladder filled with pus and surrounded by adherent

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omentum. It must be borne in mind that in the majority of cases of suppurative inflammation of the gall-bladder with calculi, the gall-bladder has been altered by repeated attacks of chronic inflammation; its walls then become thickened and contracted. I have removed a gall-bladder which was half an inch thick posteriorly. No tumour can, therefore, be felt, the bladder being shrunken and retracted well beneath the margin of the liver.

Empyema of the gall-bladder is almost invariably due to calculi. Occasionally it is a sequela of enteric fever, but it may follow simple catarrh. I removed one gall-bladder for empyema from a woman, æt. 59 years, who had suffered for three years with attacks of colic, some of which were followed by jaundice. The mucous membrane was thickened and ulcerated, and covered with a layer of soft calculous matter, but it contained no stones. I have seen cases of distinct suppuration in the gall-bladder, and these are by no means uncommon, in which the usual symptoms are entirely absent. There is no pyrexia, or it is slight, little tenderness, and only a sense of discomfort or, perhaps, hepatic colic may be present. A counterpart of these is found in some instances of appendix abscess, which forms without fever and little or no pain or tenderness. These masked symptoms are probably due to a peculiarity of the colon bacillus, which varies greatly in virulence in different cases.

Acute phlegmonous inflammation of the gall-bladder.—This only differs from the above in that the gall-bladder is attacked by a more virulent micro-organism, often a mixed infection of *B. coli* and streptococci. The symptoms come on suddenly, are very acute, and their progress is rapid. There is urgent vomiting, distinct pyrexia, well-marked local tenderness and rigidity, followed by distension of the abdomen. Hyperæsthesia of the skin, above and to the right of the umbilicus, may be present, but if the gall-bladder becomes gangrenous this subsequently disappears. Cases of phlegmonous cholecystitis not infrequently simulate intestinal obstruction, prominent symptoms being urgent vomiting and abdominal distension, with constipation. Intestinal obstruction may actually exist, owing to paresis of the hepatic flexure of the colon from local peritonitis. Occasionally extremely acute cases of cholecystitis are met with in which the inflammation is immediately followed by gangrene of the gall-bladder and general peritonitis. The symptoms accompanying the onset of these cases are similar to those of all acute "abdominal crises," such as perforations of stomach and duodenum and fulminating appendicitis. If seen sufficiently early the locality of the pain, tenderness, and rigidity, will enable a diagnosis to be made. The antecedent history may also give assistance. The following is a case in point:

A woman, æt. 67 years, was admitted under my care at the London Hospital with vomiting, constipation, and fulness of the abdomen. The previous day she had been suddenly attacked with abdominal pain and vomiting and the bowels had not acted since. The pain was at first referred to the right hypochondrium, but later to the hypogastrium. The gall-bladder region was not tender. A Riedel's lobe existed. Acute gall-bladder trouble was suspected on account of the locality of the pain. Laparotomy was performed. The abdomen contained bile-stained sero-purulent fluid, and a pin-hole aperture was found in the gall-bladder, near its attachment to the liver. The bladder contained one large stone imbedded in a pit in the liver substance near the perforation. The gall-bladder was removed and the peritoneal cavity drained.

A suppurating gall-bladder may give way and form a localised abscess which may find its way through the abdominal wall. More often it passes along the round ligament and points at the umbilicus. Ulceration may also take place into the colon, duodenum, and pyloric end of the stomach, and thus the stones may be discharged.

Diagnosis of enlargements of the gall-bladder.—The difficulties in distinguishing enlargements of the gall-bladder from other abdominal swellings are much enhanced by the fact that when the gall-bladder becomes gradually distended it frequently draws down with it a tongue-shaped process of liver which almost completely covers it. This is known as Riedel's lobe. Further, a Riedel's lobe is sometimes produced in females from tight lacing and independently of the gall-bladder. The lower edge of the right lobe projects downwards as a thin, tongue-shaped process with a depression at its junction with the liver corresponding with the costal margin. A Riedel's lobe is, therefore, by no means pathognomonic of an enlarged gall-bladder. When it is thus associated, the fundus of the gall-

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bladder often cannot be felt. A Riedel's lobe may give rise to difficulty in a case of movable kidney of moderate extent. The kidney when pressed downwards still lies under and appears to be continuous with the thin edge of liver, which moves up and down with respiration, and the swelling feels as if it occupied the front part of the abdomen and was continuous with the liver. Such difficulties usually disappear when the patient is placed under an anæsthetic.

loin; its movements are vertical, not lateral. The hand can usually be got above it and it returns to its normal position with a distinct slip.

A tumour of the kidney or adrenal is also recognised by being felt more distinctly in the loin, by its shape, by being less influenced by respiration than the gall-bladder, and by the relation of the colon. If the colon be distended with air, it covers a renal tumour, whereas it presses the gall-bladder upwards provided this viscus is not adherent. On

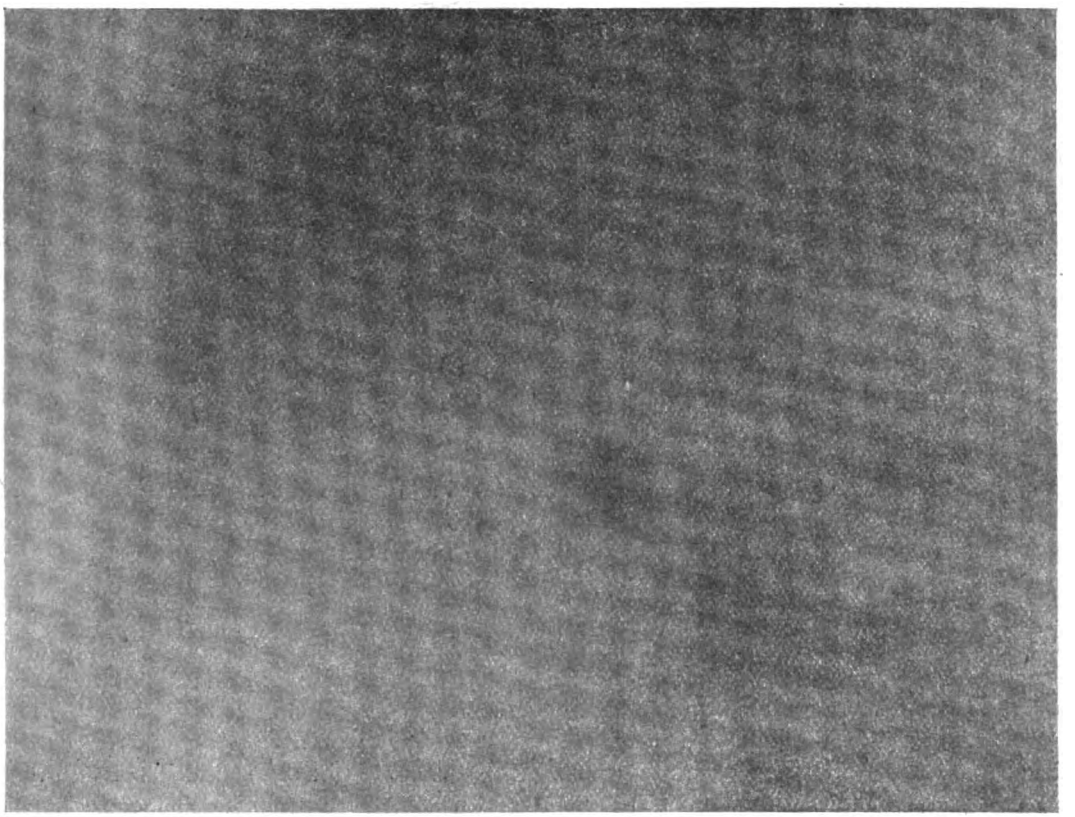

FIG. 1.—Radiograph of a calculus in the common bile-duct. The right side of the patient as viewed from in front.

The diagnostic features of a distended gall-bladder are its shape, position, the fact that it can be moved freely from side to side but is fixed above, that it moves with respiration, is dull to light percussion, the loss of resonance being continuous with the liver dulness.

Kehr draws attention to the point that when the flat of the hand is laid above the tumour a groove can be felt between it and the liver which moves on respiration.

A movable kidney is felt more distinctly in the

one occasion I met with a difficulty in differentiating a cyst of the head of the pancreas from acute cholecystitis. I was asked to see a young man suffering from enteric fever who had developed a tumour in the right hypochondrium. Although he was in the fifth week of the fever, his temperature still continued high. A swelling was continuous with the liver dulness but did not move in respiration. Under the circumstances the presumption was that it was a case of enteric empyema of the gall-bladder, which was adherent.

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Laparotomy, with drainage of the swelling, showed, however, that it was a cyst of the head of the pancreas.

Cholecsytitis and hepatic colic may usually be distinguished from appendicitis, renal colic, and gastric troubles by ordinary diagnostic methods. But serious difficulties arise in exceptional cases, of which I will cite some examples.

A young officer had been invalided from India after a severe attack of abdominal inflammation, which had been diagnosed as either hepatic abscess or appendicitis. The pain and tenderness

FIG. 2.—The stone. Actual size.

had been entirely localised in the right hypochondrium. On operation I found a very long appendix, of which the inflamed tip was adherent just below the edge of the right lobe of the liver. Again, a man, æt. 50 years, was admitted under my care with a diagnosis of acute empyema of the gall-bladder, made by a very competent physician. A fortnight previous he had been seized with very acute pain in the right hypochondrium. The pain remained continuous, and he was feverish and very ill. There was an ill-defined swelling on the outer side of the right hypochondrium, continuous above with the liver dulness, but not moving with respiration. The swelling was situated somewhat far out for the gall-bladder, but not further than I have met with it when acutely inflamed and adherent. The right iliac fossa was free and not tender. At the operation an appendix abscess was found pointing on the outer side of the hepatic flexure of the colon. The abscess extended down behind the colon and in it the gangrenous remains of the appendix were found. The differentiation between acute pancreatitis and cholecystitis may also present great difficulties, especially in obese patients. Thus I was asked to see a woman, æt. 30 years, under the care of my colleague, Dr. Dawson. Fourteen days previously she had been laid up with sudden epigastric pain and persistent vomiting. Two days later she became jaundiced. There was irregular fever up to 101°, pulse 140. "The liver was enlarged and tender, but the gall-bladder could not be made out, and there was no tenderness except over the hepatic area."

Three days before death a tender lump could be felt in the left side of the abdomen. At the autopsy fat necrosis and hæmorrhages were found in the omentum, with some local peritonitis about it and fat necrosis in the pancreas. The biliary apparatus was natural and the common bile-duct patent. An exploratory laparotomy was not performed as the indications were doubtful and the patient's condition too grave to admit of it.

Skiagraphy may be of assistance in the diagnosis of gall-stones, although it is rarely that a shadow can be obtained. Mr. Harnack has succeeded in photographing calculi in two of my cases at the London Hospital, also in one other case. The accompanying plate shows a fair-sized calculus in the common bile-duct; it is situated opposite the second lumbar vertebra, and nearer the spine than

FIG. 3.—A section of the stone.

a renal calculus. Mr. Candy kindly analysed the calculus, and found a considerable percentage of carbonate of lime. Possibly for this reason it gave a shadow (see Figs. 1, 2, 3).

Calculi in common duct.—The stones are usually two or three in number, sometimes single. They are commonly of moderate size, but I have found the duct filled with great numbers of small seed-like calculi. They may be situated at any part of the duct, but are rather more common in the supra-

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duodenal portion; there they attain a larger size than elsewhere. The cases fall into two groups—(1) chronic calculous obstruction which is incomplete, (2) acute calculous obstruction which is complete.

(1) *Chronic calculous obstruction.*—In order to understand the symptoms accompanying this condition it is necessary to consider what occurs when a stone occupies the common duct. The duct becomes greatly dilated above the stone, which does not completely occlude the duct. Bile is able to percolate around the stone, so that jaundice is not complete. The duct being in direct communication with the intestine, its contents are practically always infected by bacteria. From time to time the stone becomes impacted in the narrower portion of the duct below its usual position, and acts as a ball-valve; the impaction is probably intensified by spasmodic contraction of the duct. This accident is accompanied by deepening of the jaundice, the damming back under pressure of bile containing septic germs in the ducts above, and septic absorption, with its attendant symptoms. The life of a person with a calculus in the common duct is rendered unendurable by the recurrence of attacks of colic with incomplete jaundice. The pain is felt in the epigastrium and right hypochondrium and extends to the back and angle of the right scapula. This is more definitely the case according to Kehr than in calculus in the gall-bladder. The pain may be intense, doubling the patient up, and the vomiting urgent. The onset of the attack is followed by a sudden rise of temperature to 102°–104° F. with chills or a distinct rigor, followed by sweating. These symptoms are due to cholangitis, set up by the impaction. The chills or rigors are often repeated and in some cases constantly recurring. The symptoms of hepatic colic may date back many years. I have operated on one case with a history of fifteen years' and another of seven or eight years' duration. Chills, with moderate pyrexia and sweating, discomfort in the hypogastrium, and slight icterus, may be the only symptoms. Such cases have been mistaken for ague. Jaundice appears with the attack, gradually deepens, and passes off when relief occurs. The stools are pale and pasty but not absolutely free from bile. Vomiting is usually present, may be very urgent, and does not relieve the pain. Tenderness may exist at the biliary point, but is not so marked as in gall-bladder calculus. The severity, frequency, and duration of the attacks present wide variations. Intermittent jaundice is a marked characteristic of calculous obstruction of the common duct. In the early stages of the disease the liver gradually enlarges, in the later it diminishes. Stones may be passed into the intestine with complete relief to the symptoms. It is of extreme importance to note that the gall-bladder is practically never palpable. This, with the intermittent jaundice and chills, distinguishes calculus in the common duct from obstruction to the duct by carcinoma of the pancreas, carcinoma of the ducts, or severe chronic pancreatitis. In these latter conditions the gall-bladder is distended with bile, the jaundice persistent, complete, or gradually deepening. Exceptions to the rule that in obstruction by calculus in the common duct the gall-bladder is not distended are found in cases of associated cholecystitis, with or without calculi in the gall-bladder. Kehr states that the gall-bladder may be distended when a calculus is impacted in the duodenal papilla provided the gall-bladder has not previously been thickened by inflammation. In calculous obstruction there is progressive loss of weight, increasing feebleness and anæmia, a hæmorrhagic tendency being ultimately established. These facts must be borne in mind in distinguishing this condition from cancer.

(2) *Acute calculous obstruction of common duct.*—When a stone first enters the common duct it frequently gives rise to complete obstruction of the canal. This may sometimes be caused by a stone becoming firmly impacted in the duodenal papilla. The distinguishing feature of complete or acute obstruction of the common duct is complete jaundice of sudden onset, without distension of the gall-bladder. The absence of enlargement of the gall-bladder, the sudden onset of jaundice, with pain and colic, may enable the condition to be distinguished from obstructive jaundice, due to malignant tumour. Kehr states that acute obstruction of the common duct by calculus is associated with colic, vomiting and frequent chills and rigors. Moynihan, however, writes that symptoms of septic infection are entirely absent. After a time the impacted stone becomes loosened and may pass into the duodenum or may remain, and cause the usual symptoms of chronic obstruction.

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In the following case a stone in the common bile-duct was so firmly impacted as to cause ulceration and perforation of the duct.

Miss H—, æt. 30 years, was attacked with hepatic colic, followed by jaundice, ten days before I saw her. A rigor occurred on the eighth day, but was not repeated. There was considerable pyrexia. She was deeply jaundiced; there was rigidity of the upper part of the rectus, and a tender, dull swelling in the region of the gall-bladder. On opening the abdomen adhesions were found between the under-surface of the liver and the gastro-hepatic omentum. On gently separating these a collection of pus was found; this, being swabbed away, was followed by an escape of bile. Further inspection showed an ulcerated opening in the common bile-duct. This was enlarged and a stone the size of a small marble removed. A drainage-tube was sewn into the duct with catgut and the patient made a good recovery.

The diagnosis between obstruction of the common bile-duct due to stone, from the obstructive jaundice caused by enlargement of the head of the pancreas, from carcinoma, or chronic indurative pancreatitis, is often one of the greatest difficulty.

The cardinal symptoms of obstruction by tumour have been already alluded to as distension of the gall-bladder, intense and gradually increasing jaundice, and the absence of pain, colic, and fever usually accompanying obstruction by stone. So-called typical cases are, unfortunately, not infrequently the exceptions that prove the rule. Difficulties may arise in calculous obstruction of the common duct, owing to the presence of a distended gall-bladder from the co-existence of stones blocking the cystic duct with cholecystitis. On the other hand, the symptoms of carcinoma of the pancreas may closely resemble those produced by calculus. Thus in a man, æt 42 years, with cancer of the head of the pancreas, the symptoms began with chills, pain in the upper abdomen going through to the back, vomiting, followed by slight jaundice. Later the jaundice increased and the gall-bladder became distended.

In another patient, a woman æt 60 years, her illness commenced ten weeks previously with pain in the epigastrium, vomiting, followed fourteen days later by jaundice, which persisted. She had variable pyrexia, the temperature ranging from normal to 101°–103° F. There was an ill-defined swelling in the hypochondrium which turned out to be a distended gall-bladder.

A third patient, a man æt 41 years, with chronic pancreatitis, had had four distinct attacks of severe pain, referred to the epigastrium, vomiting, and jaundice, each lasting three to five weeks. The four distinct attacks extended over a period from December, 1899, to February, 1902. The gall-bladder was natural, but the pylorus was thickened, probably from an old ulceration.

Possibly Cammidge's test might have assisted in the diagnosis of these cases, but we did not find his earlier method reliable at the London Hospital.

It is not my intention to deal with the palliative treatment of gall-stones, which is for the most part unsatisfactory, nor shall I attempt to describe the technique of the various operations. I, of course, recognise the great value of the Mayo Robson incision and position in operations on the gall-bladder. It is often not necessary to divide the fibres of the rectus to any considerable extent transversely, as the muscle can be pulled aside if its sheath with the skin and the peritoneum is divided sufficiently extensively.

Cases of dropsy of the gall-bladder should be operated on even although there are no symptoms, for at any time infection followed by suppuration may occur.

It is my opinion that, the general condition of the patient being favourable, all cases of cholelithiasis should be operated on as soon as the diagnosis is sufficiently clear. Cholecystotomy, followed by drainage, is the operation of choice in the majority of cases of cholelithiasis.

There are, however, many cases in which I remove the gall-bladder, and consider the following to be the principal indications: when it is contracted and thickened, or when it is considerably dilated; when the mucous membrane is ulcerated or covered with calculous deposit; in cases of empyema, especially if the walls are acutely inflamed and softened; when it is perforated or gangrenous; when there is a stricture or obliteration of the cystic duct.

When a stone is impacted in the cystic duct, and cannot be removed through the gall-bladder, I prefer to remove the gall-bladder rather than to excise the stone and suture the duct; the latter proceeding must necessarily be attended by a risk of subsequent stricture.

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The necessity of removing the gall-bladder in some of the conditions I have mentioned is emphasised by cases in which trouble recurs after the operation of cholecystotomy. The three following have come under my own observation:

I performed cholecystotomy, followed by drainage, on a woman with gall-stones. About a year later she returned with a cystic swelling beneath the lower part of the scar, and complained of return of her pain. A subsequent operation revealed the fact that the fundus of the gall-bladder was dilated into a cyst containing mucus, while its upper part had become obliterated. The gall-bladder was removed.

Again, in the case of a man with cholecystitis, I opened the gall-bladder, removed the stones, and drained in the usual way. He made a complete recovery, but came back a few months later complaining of return of his pain. It was subsequently necessary to remove the gall-bladder, which was found shrunken and imbedded in adhesions.

A third case was a lady, æt. 65 years, who five years previously had been operated on elsewhere for calculi in the gall-bladder. A year after this operation a swelling appeared beneath the scar. When I saw her she had an enormously dilated gall-bladder. I operated, and with much difficulty dissected out the gall-bladder, which was adherent to the colon, stomach, omentum, and parietes. The cystic duct was completely obliterated and calcified.

In the majority of cases of cholecystectomy the abdominal wound can be closed, but in all cases where septic infection of the gall-bladder is at all acute, drainage should be performed by a tube placed in the junction of the cystic with the common ducts, as recommended by Mr. Moynihan. Cholangitis is tolerably sure to co-exist, and, unless the hepatic duct is drained, may spread into the liver, causing multiple abscesses and pyæmia, or death from septic absorption due to cholangitis.

A careful examination of the common and hepatic ducts for stones should be made in all operations for stones in the gall-bladder. This is especially required if cholecystectomy is contemplated, for should there be an irremediable narrowing of the duct, a means of diverting the flow of bile by way of the gall-bladder into the duodenum will be lost if the gall-bladder has been removed.

In 1904 I reported three successful cases of cholecystectomy, and since then I have performed the operation seven times, with one death. The fatal case occurred in a patient with acute cholecystitis due to a stone impacted in the cystic duct, with softening amounting almost to a gangrenous condition of the duct. She died with pneumonia.

It is needless to say that all cases of calculus in the common duct should be operated on. Septic infection, either manifest or latent, exists in all; if latent, this is due to the absence of retention of bile for the moment, or to a low degree of virulence of the infective agent.

Even in chronic cases there is progressive emaciation and a hæmorrhagic condition will ultimately be brought about.

My experience of the use of chloride of calcium to prevent hæmorrhage in these cases of chronic biliary obstruction with jaundice has been favourable.

Much discussion has taken place among surgeons in regard to the question of drainage after choledochotomy. By some of the best authorities it is laid down that drainage is essential after all cases of choledochotomy.

I have performed choledochotomy seven times, in each case successfully. Twice I have drained through the gall-bladder and once through the common duct. Four times both the wound in the duct and in the parietes have been closed.

My impression is that drainage need only be used in cases with well-marked chills and fever, and that we may exercise the same discretion in employing drainage as in appendix operations, but that in cases of doubt it is better to decide in favour of drainage as the safer course.

April 30th, 1906.

Messrs. Archibald Constable & Co. will very shortly publish 'Recent Advances in the Physiology of Digestion,' by Professor E. H. Starling, M.D., F.R.S., Jodrell Professor of Physiology. The book contains illustrations and diagrams. The author expresses a hope in his preface that the volume may serve to interest a large audience of students and medical men in the growing importance of these subjects, and that it may give them an idea of the aims and objects of this particular branch of physiological research.

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ON THE NON-OPERATIVE METHODS OF TREATING CHRONIC DISCHARGE FROM THE MIDDLE EAR.*

By RICHARD LAKE, F.R.C.S.,
Surgeon Royal Ear Hospital.

GENTLEMEN,—The subject I have chosen for this lecture I believe to be of great interest to every medical man engaged in general practice. At least a century ago, when otology was first beginning to establish for itself the right to be considered a separate department of surgery, the best writers on the subject, and those who took the greatest interest in the development of the then young speciality, complained greatly that medical men commonly advised their patients not to have anything done to an ear if it discharged, for the two following reasons. The first was that, if it occurred in a child the discharge would probably cease about puberty or early adolescence; and the second that in all cases, if the discharge was checked, especially if it was checked suddenly, it might very probably cause the death of the patient. And you may remember that Francis II of France died at the age of seventeen from cerebral abscess, and that his physician explained the profuse discharge from his ears on the theory that, as his nose was obstructed, the noxious humours had to find their outlet by that route. I am sorry to say that there is still, amongst the laity, especially of the poorer class, a tendency to object to any checking of an otorrhœa, though I am also extremely glad to say that year by year this tendency becomes less. The origin of this theory was undoubtedly due to the fact that, when an intracranial lesion occurred the temperature rose suddenly to a high level, and temporarily checked the discharge from the ear, and at the then state of medical science the effect was confused with the cause. My first clear recollection of anything medical, in my early hospital days, was that one day we were taken into a small ward where there was a patient in a state of wild delirium, suffering from meningitis, the result of a neglected otorrhœa, and I remember my horror on hearing from the mouth of the physician the words: "Gentlemen, we can do nothing for these cases." I am thankful to say that such a remark would rarely be heard nowadays, and that is just five and twenty years ago, though cases do occur where assistance is called in too late; this is more often the fault of the patient rather than the fault of the medical man.

Such a case as this occurred to me a few years ago. The patient was a young man, who had complained of headache, and stopped away from his work for two days. The doctor had not seen him until the third day, and was unable to formulate immediately any definite diagnosis, though he discovered that there had been a one-sided otorrhœa from childhood. The next morning, however, he considered that the symptoms were sufficient to warrant his considering it to be some form of intra-cranial lesion, secondary to the discharge from the ear. When I saw the man the same afternoon, he had dilated stabile pupils, nystagmus, rapid pulse, and temperature of 103°. He was obviously suffering from meningitis and probably some other intra-cranial lesion. We operated immediately, and evacuated two extra-dural abscesses, cleaned out a large amount of septic clot from the lateral sinus, but found, unfortunately, that our patient was also suffering from general meningitis, and although consciousness was restored, he died three days later.

This is an example of a fatal case. Now, to give an instance of the importance at present attached to discharge from the ear in contradistinction to that obtaining twenty or thirty years ago, we must notice the fact that few insurance offices will accept a proposal from a man suffering from this trouble. One office, with which I am well acquainted, objected strongly to inserting any question about the ear in their medical report. However, they quickly changed their minds when a man who had been accepted in their office, but, fortunately for them, had not paid his premium, died within two months from cerebral abscess, having been accepted as a first-class life in another office *aware* of the diseased ear.

It therefore behoves us as medical men to look upon all discharge from the ear in the same way as one looks upon a fire near to a store of combustibles, which might be ignited at any moment by a chance spark. The patient is never safe, and we do not know the proportion of cases in which

* Delivered before the Brixton Medical Society.

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serious complications occur, but we do know that at no period of life is the patient who suffers from an otorrhœa immune. There are also less serious results, relief from which is greatly appreciated both by the patients and their friends. For instance, the capacity for intellectual employment is considerably increased when a source of irritation so close to the brain is removed, and I have twice known the character of patients completely changed after the cure of a serious aural lesion. In both instances the patients had been previously morose and irritable.

I am going to ask you to consider that chronic discharge from the middle ear be defined as cases in which the discharge has existed for six weeks and upwards. Before considering the treatment at all, it is well that we should *briefly* consider the preliminary investigation and diagnosis; for it is necessary, or at any rate advisable, that we know the extent of the mischief, the situation of the perforation, and any other information, such as the presence or absence of aural polypi, before we commence our treatment.

It is therefore necessary that the ear be thoroughly cleansed and equally thoroughly dried. Every fragment of loose skin and every little piece of inspissated discharge must be removed from the meatus, for the aural passage is already sufficiently small, and we cannot make an accurate diagnosis as long as there is any foreign matter within the canal or the least suspicion of moisture. As a general rule it is not advisable to syringe the ear before examining it, although, if other methods fail to cleanse the ear, we must have recourse to syringing. The reason for this is that the temperature of the lotion that it is necessary to use causes great hyperæmia of the parts, and alters their appearance so materially that several minor mistakes are liable to occur, such as considering a patch of exposed mucous membrane to be an aural polypus. The best method of cleansing the ear is to mop it out with small pieces of antiseptic cotton-wool, rolled up fairly tightly in the fingers, introducing them by means of a pair of forceps. There is no special pair of forceps which is better than any other; the forceps I am in the habit of employing are those of Professor Lucae. It is, of course, necessary that one's hands should be cleansed before this is commenced. As one gets into the deeper parts of the canal, the pieces of wool should be passed through a speculum, in order that they may be guided in various directions. If there are small patches which it is difficult to dry in this way, a small piece of cotton-wool may be wrapped round a probe, roughened at the end, and the ear mopped out. In wrapping wool round this small metal probe, one must be careful that the wool projects a good long way beyond the metal, otherwise pain or injury may be caused.

Of course, in examining the ear a reflected light must be used, and the speculum inserted while the ear is drawn upwards and backwards with the hand not holding the speculum. When the speculum is introduced into the ear, the auricle is grasped only by the first finger and thumb, and the speculum is held in place by two of the remaining fingers; or the auricle may be held up between the little and the next finger, and the finger and thumb manipulate the speculum.

We may divide our discharging ears into the following subdivisions, and I wish to include in these subdivisions only those ears in which the discharge is excreted in the cavity of the tympanum, or one of its accessory parts. Thus, we may have pus or purulent fluid coming through a small, a medium-sized, or a very large perforation, or we may have it exuding from a perforation in Shrapnell's membrane, or a sinus in the bone by the side of the membrane, the membrane itself being intact. It is very essential also, in considering the treatment, that the site and size of the perforation be taken into consideration. If the perforation is a very small one, it must be perfectly obvious to anyone who considers the matter that, however much one may syringe down the external meatus, only an extremely small amount of the irrigated antiseptic fluid can possibly gain admission into the cavum tympani, or cavity of the middle ear, so, in a direct ratio, the larger the perforation, the more easily can it be brought under the influence of injections. Again, if the perforation is in Shrapnell's membrane, or if the discharge comes through a small sinus leading into one of the cells connected with the antrum or attic, it seems to me that it must be obvious, even to a person of medium intelligence, that irrigating the canal alone will be without direct influence on the disease, though indirect results are obtained even by this means; and in these cases, as there is no longer a collection of septic and decomposing matter in the

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external auditory canal, the cavity may cease to excrete pus, and be enabled to dry up simply by the absence of re-infection from without.

We must not, however, make light of the difficulties attendant upon a correct diagnosis in these cases, for a correct diagnosis, as I have said, includes many minor details beyond the size and situation of the perforation. Pulsation of the pus is never a good sign, though it is not really one of any great importance; it is supposed to be caused by an enlargement of the small vessels in the tissues, and this is probably correct. It is only by careful study and considerable experience that one can appreciate the subtle changes which have occurred in the drum and deep parts of the meatus.

We now will rapidly consider the means we have at our disposal for combating this disease, which offers so many barriers to the remedies applied by the surgeon, whose course is usually tedious, and which tends so easily to cause both the practitioner and the patient to become impatient and to lose interest.

We may take it that the most usual method, and the one most in favour, as well as frequently the most suitable, is antiseptic irrigation. After this follows insufflation of powders, next the use of guttæ, or drops, and finally surgical interference.

With regard to irrigation we have, we may say, hundreds of solutions, but a dozen will be all that we require, and to select the best is impossible; but all antiseptics that are used should be of sufficient strength, not only to kill the germs, or such germs as they are able to come in contact with, but also to inhibit sporiolation. Of these, lysol, izal, and creolin are more or less alkaline, and on account of their alkalinity are able to saponify the fatty matter present in the discharge and on the sides of the canal, and so obtain greater cleansing. Perchloride and biniodide of mercury, carbolic acid, with formalin, are strong antiseptics. Boracic acid is an antiseptic and slight astringent.

One great point to remember in the use of any lotion is that it is better to use a glass syringe one's self, and always advisable that the patients themselves use a glass syringe, over the nozzle of which is drawn a piece of fine indiarubber tubing. Glass is cleanly, and any particular part can be seen. The rubber tube is passed gently down as far as it will go into the canal, and serves two purposes. First, it causes no pain, and therefore is extremely useful in children; and secondly, it gets beyond the discharge, so that with much less force a greater clearing out is effected, and I should strongly advise all those who are not in the habit of using this little piece of tubing to try it and see what they think of it.

Another important thing to remember with regard to lotions is that, as a rule, lotions are not to be persisted in for more than four to six weeks if they do not appear to be doing good, but another antiseptic selected, and preferably one of a different class. Nor should one which did good at first be persisted in if the benefit be not increased. And, lastly, in order to obtain the best possible results one should either perform one's own syringing or be quite certain that the syringing is done in an efficient and satisfactory manner; for I am perfectly convinced that hundreds of cases which are now operated upon would rapidly and assuredly recover if the irrigation was done according to the lines I have laid down. I have often seen the most extraordinary differences when the syringing has suddenly been made effective. The patient should always be seated; the fluid should be of a comfortable temperature, of about 100°, and where possible the ear should be thoroughly dried after each irrigation. Sometimes, after irrigating an ear and cleansing it, especially if the membrane is nearly destroyed, insufflation of sterilised boracic powder may be combined with irrigation. Otherwise the more frequently irrigation is performed the better, but the minimum should be twice a day. The number of times may be diminished according as the discharge diminishes, but should be increased if the discharge is excessive. I have sometimes thought that a $\frac{1}{4}$ to $\frac{1}{2}$ per cent. solution of formalin acted particularly well when the discharge was very purulent, and the mercurial solutions when there was a large amount of mucopus and mucus being secreted. This, however, is very likely only fancy.

The next means at our disposal is that of insufflating powders. There are several methods of insufflation, either by using a powder-blower (Macbraith's, Maw & Co.), or by taking a small piece of quill or glass tubing with smooth ends, and about a foot of rubber tubing, such as is used for feeding-bottles. The necessary amount of powder, which must not be more than grs. iii–v, should be put into the glass tube, or quill, which is inserted into the external meatus, and then the free end of the

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rubber tube is carried to the mouth and the powder blown out. Now, it must be thoroughly understood by all one's patients that no insufflation of powder must ever take place into a dirty ear. The ear must be thoroughly cleansed and dried by the surgeon before the first insufflation, and if the discharge is large in amount, but the surgeon still wishes to use insufflation, it must be combined with irrigation. *Pari passu* with the diminution of the discharge the frequency of irrigation may be diminished until it is used only twice a week, or even less, but in these cases it is well that the irrigation is sometimes done the day before the surgeon is to be seen.

Boracic acid is probably the best powder that we have at our disposal. Next comes iodoform, which may be rendered inodorous, as you know, by the addition of a few drops of geranium oil.

Under no consideration whatever should powders be used in any cases where the perforation is in Shrapnell's membrane or in the drum itself, small, or situated high up. Therefore the chief indication for the employment of this method is—total destruction of the membrane, or a large perforation situated low down. If the perforation is small and situated high up, the use of any powder is not unattended with danger, as it might easily occlude the perforation, and the pent-up discharge might thereby be diverted into channels less safe than the external auditory canal. In this connection I would remind you of the celebrated Parisian quack aurist, who for a short time had a tremendous reputation for curing discharging ears by means of a white powder, till a run of bad luck set in and a lot of his patients died, and they died from intracranial lesions. An inquiry was held, and the powder was found to be plaster-of-Paris.

Drops are the next means of dealing with these matters, or, more correctly speaking, lotions used as drops, and they are more generally applicable in suppurative disease of the middle ear than in any other form of disease, and may be combined with both irrigation and insufflation, and brilliant results will often attend their judicious employment. They are especially valuable under the following circumstances. Alcoholic drops if a discharge is watery and extremely rebellious to treatment, where the mucous membrane is fleshy or there is a tendency to the formation of polypi; peroxide in children, where the discharge is thick, the perforation small, or situated high up, in Shrapnell's membrane, or when a sinus outside the tympanum is present. When there is a large perforation and pus is coming down from the attic, and when cholesteatoma is suspected in that region, one should, if possible, use the alkaline form of peroxide, as it is less irritating. Ten or twenty drops should be instilled into the ear, the head laid on the opposite side, and the head should remain on that side for twenty minutes. If alcohol is used it is well to begin with a low grade of strength, and to increase it; the same with peroxide. In connection with peroxide it is advisable to remember that sometimes the amount of gas evolved may give rise to vertigo if the external semicircular canal is exposed. Alcohol may be employed as a vehicle to carry antiseptics, such as carbolic acid, boracic acid, or mercury. Again, dilute hydrochloric acid may be used in the same way to remove small pieces of necrosed bone, which might otherwise be difficult to eradicate without operation.

Briefly, as auxiliary forces we can occasionally bring in to aid us in obstinate and difficult cases, especially if the perforation be small or situated in Shrapnell's membrane, a method of washing through the ear from within. An Eustachian catheter is passed and proved to be in position, and the nozzle of the syringe is placed in the end of the catheter and the ear syringed out with a weak boracic acid solution or a sterilised normal saline; and in the case of attic perforations it is often well to wash them out by means of a Hartmann's cannula, using a strong antiseptic in the little bag which is usually attached.

Although in these remarks which I have made to you this evening I have attempted to deal with the treatment of middle-ear suppuration I have carefully abstained from more than a passing reference to operation. The conditions which should cause one to undertake operative interference are practically the following: if an acute case suddenly becomes active; where, in children, the removal of adenoid vegetations has failed, and the disease has lasted for a considerable period—say six months—under good treatment; and in adults, without the adenoid operation; where there is giddiness and pain, with recurrent attacks, facial paralysis, incurable cholesteatoma, lateral vertigo on syringing, persistent pain, contraction of the external meatus, fistulæ which will not heal,

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necrosis, and tubercle. Beyond this, I should advise that one remember that the constant absorption of septic products may act so deleteriously on the patient's health as to justify operation. I do not think that it is justifiable to operate unless one has one of two reasons for operating: either the case is incurable without operation, or it is necessary for the patient's life or health that operation should be done.

Finally, as I say, in children never consider that all has been attempted unless one has removed the adenoids efficiently. That alone in children will cure over 50 per cent. of the cases. And, finally, let me urge the necessity of keeping a careful watch on any patient suffering from otitis media. In that state the patient is uninsurable in any respectable life office, and when a patient has exhausted every other means of cure except operation and obtained no relief, at any rate it is our duty, both to ourselves and our profession, to place the matter before him in such a way that there is no doubt but that he understands the gravity of the situation.

April 30th, 1906.

Alimentary Treatment of Bile Fistula.—Gross has described his experience with a patient who had a profuse flow of bile through a fistula resulting from a cholecystostomy. After several weeks of this loss of bile Gross checked it by instituting the "alimentary course of treatment" recommended by Jaboulay and Patel. They found that the flow of bile could be checked and the healing of a fistula promoted by preventing the accumulation of bile in the natural reservoir for it, the gall-bladder. This they accomplished by not leaving a long interval between meals. The meals were given at three-hour intervals, the patient being waked to eat twice between midnight and dawn. Gross ordered his patient to eat every three hours, night and day, and in twenty-four hours the amount of bile escaping by the fistula had been reduced by one half. By the end of the ninth day scarcely any secretion escaped through the fistula, and five days later the flow had entirely ceased and the fistula soon healed over. The immediate success in this case, after weeks of persistence of the flow of bile, was too striking to be merely a coincidence. *Journ. A. M. A.*, vol. xlvi, No. 14.

WITH DR. SAMUEL WEST IN THE WARDS OF ST. BARTHOLOMEW'S HOSPITAL.

HERE is a woman whose case is of some little interest. There is extreme irregularity of the heart now, but there has been no definite history of any heart attack of any kind until on December 4th she had a bad attack of diarrhœa and vomiting, which was followed almost immediately by pain, palpitation, and shortness of breath. On admission she had an extraordinary irregularity of the heart's action, and the slightest effort brought on palpitation. She has improved a little, and will, no doubt, get much better. The diarrhœa did not, of course, produce a heart lesion, though it did lead to heart symptoms. This is an illustration of what occurs so often in heart disease. The patients get along very well in spite of the disease, until they get ill from some cause or another, not cardiac, and then cardiac symptoms appear. This is seen in all forms of cardiac disease, but especially in affections of the left ventricle. A patient, for instance, has aortic disease but is said to be quite well. Then all at once he gets a strain, runs to catch a train, has worry and cannot sleep well, gets an illness such as a typhoid fever, or, as in this case, only an attack of diarrhœa, and then he gets heart symptoms.

There is no evidence of any valvular lesion in this case. Of course it does not follow that there is nothing the matter with the heart because there is not any murmur heard, for the patient may have the worst of all diseases, namely some affection of the heart-muscle. There are three or four patients in the wards now who have weak hearts, causing shortness of breath, palpitation, and pain, so that they are practically incapacitated, yet physical signs give no evidence of gross disease except an increased area of cardiac dulness.

Again, a patient may have disease in the anatomical sense, *i. e.* cardiac lesion, and yet have no discomfort. And then some day, from one cause or another, symptoms appear, and they have discomfort, dis-ease as well as disease. Till then they may not believe they have anything serious the matter because they have no discomfort, and they date their disease from the onset of symptoms,

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though the doctor may know it existed long before.

Here is another woman who presents some features of interest. I ask you first of all to look at her feet, which you notice are swollen, and that this condition extends to the middle of her body. Yet it is not œdema, for it does not pit on pressure. The patient has well-marked Graves' disease. Several interesting things have happened. When she came in the eyes were very prominent, the thyroid gland was enlarged, though not to any great degree, but there was a good deal of tremor and the patient was evidently very ill. Gradually she got very much better, but about a fortnight ago, without any apparent reason, her legs began to swell, and the swelling rapidly spread upwards to the body, and now reaches to above the waist. We have to deal with a very rare condition, the association of myxœdema with Graves' disease. Her history is that fifteen months ago she began to get thinner and to lose strength, and then her neck was noticed to be getting larger. Six months ago she had pain in the head, palpitation, and sleeplessness, and two months ago diarrhœa and vomiting. She also had a little diarrhœa the week before admission. She had been in the hospital since November 18th, and on admission there was slight œdema of the feet, but that quickly passed off. On December 8th her condition had suddenly become quite alarming. She was drowsy and stupid, and seemed to take little or no notice of what was said. She had been vomiting a good deal, and had a peculiar panting respiration, which might have passed for diabetic respiration. The urine was examined, but there was nothing abnormal found, either albumen or sugar. She remained in that serious condition for many hours. She was too ill to justify us in trying any treatment which was in the least in the nature of an experiment. Her respirations were 60-64 per minute, she had a red, flushed face, such as is seen in diabetes, her pulse was only 104, and the temperature rose to 101° F. She had been taking citrate of caffein and strychnia as general stimulants. Previously she had been given digitalis to quiet her heart. She then became so feeble that the digitalis was stopped, and adrenalin given, but that also was soon stopped. Then we gave her oxygen, and that is what, I think, did her good. It was administered continuously for many hours—fifteen, I think. Under its influence she began to rally, and the symptoms described subsided. There have been one or two threatenings of their return, but the attacks were averted by using the oxygen again and giving a sedative. I have never seen this toxic condition in Graves' disease before. When I saw her I was almost afraid she had been put upon thyroid extract, but I found it was not so. The acute onset and rapid development of myxœdema in this case reminds me of instances of acute sclerodermia, which may run over the body in the same rapid way, and it is interesting to note that in some of the accounts of Graves' disease their association with sclerodermia is actually described.

This youth was admitted with considerable swelling over the hand, wrist, and forearm. He was well until three weeks ago, when he had a chill and pain in the abdomen, but that has nothing to do with the present illness. On the morning of the day of admission he noticed that his hands were swollen and red, and this was followed by pains in the hands. He had a similar condition in one great toe. It was quite an acute swelling, and the question was as to its nature. It was suggested it was angeio-neurotic œdema. True, it was an œdema, but does the term "angeio-neurotic" carry us much farther? Many of these cases come into the erythema group. Perhaps you may say that does not help one much either, because erythema is so indefinite a term and includes so many varieties. You may recall cases in which a patient gets a sudden swelling on the face, lips, and nose, or round the eyes, which disappears in an hour or two. Sometimes it involves a larger area, perhaps the whole head and face, and such patients look as if they were in the last stage of renal disease. Or it may affect the hands. It is closely akin to urticaria, only it does not itch. Erythemas as a rule burn and tingle and are painful, and very often are hyperæsthetic, whereas urticarias itch. The two groups develop in much the same way, but urticaria is much more limited in extent. Another name has also been given to a similar condition, namely "erythromelalgia," which is a curious disease, but I do not think this is an instance of it. I saw a case in private some time ago and the only name I could give to it was erythromelalgia. The patient had tingling and

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redness of both hands, and was very much distressed by it. She was an elderly woman, as most of these patients are, and she had some atheromatous patches in her arteries, and the question arose whether the disease might not be vascular in origin. The interest about that case is that the doctor wrote and said she had ultimately developed a sort of septic arteritis, of which she died. I think it is possible she had something of that kind affecting the circulation of her limb at the time I saw her, though I could not get sufficient evidence of it.

A large number of these erythemata are due to poisons absorbed from the intestines, and if a purge be given to get the intestines clear, the symptoms disappear in the same way as they do in urticaria.

Here is a boy with a very large liver and spleen. He originally came in very ill, with a small empyema, which was opened, but the wound would not heal. He was sent away into the country for some months, and returned better, and with the sinus almost closed. Then it opened again, and there is still a little discharge. I suppose the enlargement of the liver and spleen are due to amyloid disease, but it is out of relation to the amount of discharge he has had. I thought at one time it was possible he had actinomycosis, but we have not found anything in the discharge to confirm that. Some diseases have a special tendency to produce amyloid disease, and actinomycosis is one of them, quite independently of the amount of discharge. Tubercular disease is another. I thought this boy had tubercular disease, although we have not found any tubercle bacilli in the discharge. The prognosis of amyloid disease is that of the original disease; if the disease which caused it is curable the patient may get well.

We have in this ward two cases of ascites, both having cirrhotic livers, and both of them with this dark-coloured urine which you see. When you saw this case first the ascites was acute, the muscles had not lost their tone, the abdomen was round, and the intra-abdominal tension high. Now the abdomen is flattened and has the usual bulging in the flanks, and the tension is now low. The fluid has re-accumulated, and I think there is as much there now as when he was first tapped. Yet the difference in tension is very well marked, and so is the difference in tone in the muscles.

Here is another case of the same kind. He had not so much ascites. The abdomen was not tapped and the fluid is going away. I should have thought the fact that this man has got well with simple rest was the best argument to prove that the ascites was mechanical and not toxic, because the mechanical conditions only have been altered and the fluid has gone away. The toxic theory of ascites in cirrhosis of the liver is a mischievous one, because it seems to imply that nothing can be done for it, yet many of these cases will lose the ascites entirely if they can be kept for a time at rest.

I now want to show you this morbid specimen in connection with a patient whom you saw in the ward at a previous visit. He had an extensive area of dulness at the right upper part of the chest, and greatly dilated veins, with swelling of the head and neck and right arm. Auscultation showed that no air entered the upper lobe of the right lung. Here is the new growth, at the root of the lung, surrounding the bronchus of the right upper lobe especially and of the lower lobe too to a less degree. At the base of the lung there are two large cavities, which are necrotic, but not gangrenous. Nearly the whole right lung is airless and solid, in the condition which has been called solid œdema, or, better perhaps, œdematous consolidation. In this consolidation necrosis or disintegration may occur, and produce cavities, which have been described as broken-down secondary growths, but there is no new growth in them at all: the tumour is a soft carcinoma. Some of these primary tumours of the root of the lung are very difficult to diagnose. I had a case some time ago in which at the post-mortem I was told there was no new growth; still, I felt sure there ought to be. All that could be seen was slight peribronchial thickening round the main bronchus and extending a little distance along some of the divisions. It was not until microscopical sections were cut that it was found to be carcinoma of the scirrhus variety, as is usual in most of these cases. There is very little to be found about primary root cancer of the lung in literature; but a full account of it, based on cases I have described, will be found in my book on respiratory diseases.

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INTERNATIONAL CLINICS, vols. i and ii, Fifteenth Series. (J. B. Lippincott Co., Philadelphia and London.)

The volumes of this quarterly continue to appear with great regularity, and to supply a large amount of valuable clinical matter in all branches of medicine and surgery.

The first of these volumes opens with a clinical lecture on cardiac asthma by Dr. Merklen, of Paris. There are three important elements to be considered in this affection: first, the nervous and paroxysmal dyspnœa; secondly, the mechanical dyspnœa due to pulmonary stasis, and sometimes to œdema; and, thirdly, the disturbances of the general circulation. Minor attacks of this condition are not uncommon, but the graver forms are less known, and the treatment of all forms is fully discussed by the writer. The diagnosis of heart-disease is also discussed by Dr. James J. Walsh, of New York, who lays stress on the valuable information to be obtained through the eyes and hand of the examiner, and by Dr. J. Porter Parkinson, of London, who discusses the diagnosis of heart-disease in children. There is also a valuable clinical lecture by Dr. Morris Manges, of New York, on cases of aortic stenosis and adherent pericardium. In this volume there is a striking article by Dr. Archibald Young, of Glasgow, on skin-grafting in the late treatment of severe burns involving extensive areas of skin. He describes an essential modification in the operative technique of Wolfe's or Krause's (whole thickness of skin) method, and gives illustrative cases. The accompanying photographs certainly point to very much better results than one is accustomed to meet with in such serious lesions. Although from the æsthetic point of view there is something left to be desired, from the point of view of function and the avoidance of contraction there would appear to be a great gain. This volume also contains an account of the progress of medicine during 1904, which includes medicine, surgery, and treatment, but the subject is too large for the hundred pages allotted to it, and the result is not satisfactory.

In the second volume Dr. Roger, of Paris, describes several cases of galloping typhoid fever. These proved fatal in less than a week's time, and are divided into two classes. In the first class death is due to general intoxication, the system not being able to resist the immediate effect of the typhoid poison. In the second class not only the symptoms but also the local lesions progress with great rapidity, deep ulceration being present. A new general anæsthetic, scopolamine, has been employed by Dr. Felix Terrier, of Paris. The hydrobromate of scopolamine is the salt employed, and it is administered by hypodermic injection in doses of $\frac{1}{6}\frac{1}{4}$ gram., along with $\frac{1}{6}$ gram. of morphine. Two or three injections may be required, and sometimes the inhalation of a small quantity of chloroform is also employed. We do not think this drug has been much used in England yet, and probably English anæsthetists will wait for fuller reports from the Continent before employing it.

RECENT ADVANCES IN PHYSIOLOGY AND BIO-CHEMISTRY. EDITED by LEONARD HILL, M.B., F.R.S. (Edward Arnold, London, 1906.)

PHYSIOLOGY, and especially the chemical side of it, has now come to have such an important bearing on practical medicine that it is almost essential for the physician to have some knowledge of the work that is being done in this direction. On this account a book in which are collected the main points of recent research which bear on pathology and therapeutics is very welcome, and this work will certainly give the practitioner all he requires. A perusal of the subjects dealt with will make practical work more interesting, and will suggest many possibilities of treatment on rational lines. Besides the editor, the other contributors are Benjamin Moore, J. J. R. Macleod, M. S. Pembrey, and A. P. Beddard.

THE Annual Medical Golf Tournament will this year be held on Thursday, May 31st, at Cassiobury Park, by kind permission of the West Herts Golf Club. The tournament will be played under the usual conditions, viz. match play of 18 holes *v.* Bogey, in two classes: (1) handicaps of 12 and under; (2) handicaps over 12. Foursomes will also be played for sweepstakes. Competitors must send in their names by May 30th, with lowest club handicap and 5/- entrance fee, to the Hon. Sec., Rolf Creasy, Windlesham, Surrey.

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A CASE OF EMBOLUS BLOCKING THE BIFURCATION OF THE AORTA. GANGRENE OF ONE LEG. AMPUTATION UNDER LUMBAR ANALGESIA.

By ARTHUR E. BARKER, F.R.C.S.,
Surgeon to University College Hospital.

EMBOLIC blocking of the aorta at its bifurcation appears to be a rare accident, judging from one's observation and reading. For this reason it appears desirable to report the following case:

Mrs. S—, æt. 41 years, a healthy-looking woman, but rather stout, on waking on Thursday morning, February 16th, 1906, noticed that both feet were cold and that the left was beginning to be painful. She had been walking about until the night before as usual, but had some trouble from very large varicose veins reaching up to the saphenic opening, which had existed since girlhood. Nineteen years ago she had rheumatic fever, and has suffered since from shortness of breath and giddiness and swelling of the legs after standing for long. She has also suffered every winter from cough and bronchitis, but has none at present, and last year she states that she had pleurisy.

On admission to University College Hospital on February 17th, 1906, she was well nourished, but a little yellowish and pale. She complained of numbness in the lower part of the left leg and severe pain in the upper part of the leg, which was greatly swollen. This was partly due, above the knee, to the enlargement of the veins, one of which went obliquely from outside the knee upwards and inwards, and was huge. The thigh was quite warm, the knee much cooler, and the foot stone cold. The lower part of the leg was glossy, hard, and exquisitely tender. Just in front of the internal malleolus was a bleb, three quarters of an inch in diameter, containing yellow fluid. The

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great toe was dead white in colour for the most part, but there were purple patches on the under-surface, due to blood under the skin. The dorsum showed a dark, dry patch, and also the dorsum of the foot. The under surface of the foot was purple, with dried-up patches, and the smaller toes were in the same condition. No arterial pulsation could be felt anywhere in the limb, not even in the femoral. There was no evidence of thrombosis in the veins. The lower part of the leg and foot were quite devoid of tactile sensation.

On the right side there was no sign of gangrene in leg or foot. Both were warm and sensation was good. But there was no pulsation in either tibial nor in the popliteal, and on admission there was no trace of it in the right femoral, though later it could be felt. At the bifurcation of the aorta there was considerable tenderness. The radial arteries could hardly be felt, and the pulse was very irregular. The heart was found to be much enlarged, but emphysema prevented a clear mapping out of the cardiac dulness. A presystolic murmur was found, but the tumultuous action of the heart precluded all definite description of the sounds. The action of the heart was very irregular, especially in force, and only about half the beats could be felt at the wrist. There were a few râles at the base of the lung on each side.

From all this it was quite plain that a large embolus had been arrested for a time at the bifurcation of the aorta blocking both common iliac arteries. But within a day or two it had clearly been forced further into the left, and probably by this drawn from the right, for the pulsation gradually returned on that side. The clot probably was "riding" on the bifurcation in the first instance, but reached further into the left common iliac, which, being more in the direct line of the aorta, would be most likely to catch any embolus coming from the heart. That the obstruction on this side was very complete was obvious from the rapid onset of the gangrene; on admission it was well begun, and a couple of days later, while the right leg had almost completely recovered, the left foot was black and blue and covered with blebs, and the pain became terribly severe and was only partially mitigated by large doses of opium. In addition to this the large veins all up the limb became inflamed and very soon thrombosed, which added much to the suffering, for the thigh, too, became exquisitely tender and œdematous. The temperature was now high, 102° to 103·2°, and appetite was lost. Dr. Bradford, who was kind enough to examine the heart and lungs, took the gravest view of the patient's condition and confirmed the previous examination.

The question now arose, what was to be done? It was quite evident that without amputation death must soon take place. But the general condition was so grave that it was questionable whether even this would save the woman. Sepsis had begun and had led to phlebitis and extensive thrombosis. Moreover, the patient could not lie down, and the danger of a general anæsthetic, particularly in the sitting position, had to be considered.

In the first place, however, we were compelled to wait to see how far the gangrene was going to extend upwards. This point was determined in a couple of weeks. Complete death of the tissues of the foot and leg up to the upper third had occurred by this time, as far as the eye could tell. And though there was no "line of demarcation" externally the colour of the knee and thigh above was nearly normal, except for the phlebitis. But one could not but doubt that what was visible corresponded to changes in the deeper part of the limb. It seemed more probable that the gangrene of the deeper parts extended farther than in the more superficial parts, as is so often the case. At the beginning of March it was quite clear that if any amputation was to be done at all it should not be delayed, and that it would have to be done in the middle of the thigh to get into even relatively healthy tissues.

But besides the ordinary dangers of so severe an operation there was another which had to be confronted. The enormous veins in the thigh were full of septic clots and were inflamed. It appeared quite possible that during the manipulations of amputation or the struggles during anæsthesia that some of these might be loosened and carried into the circulation, with the gravest consequences. A case in another ward of the hospital had recently died in this way, and it was suggested to me that I should first dissect out the main vein. This measure did not, however, appeal to me, as I felt it would add to the severity of the operation, and, besides, would give no guarantee that other, deeper, veins would not furnish clots.

Having decided to remove the thigh through its middle without any special attention to the veins, except the most careful protection of them from all squeezing, the question of an anæsthetic had to be determined. In this I was much influenced by the very grave opinion expressed by the physicians who had seen the case as to the condition of the heart and lungs. A general anæsthetic seemed almost certain to end fatally. And as I had had a considerable experience recently of spinal analgesia with stovaine, I elected, though with some misgivings, to perform the amputation with its aid,

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and the patient having expressed herself as perfectly willing, the injection was made in the usual way in the second lumbar interspace at 3·52, and a special member of my staff was told off to observe the effect. The following points are abstracted from his notes. In seven minutes sensation to pain was abolished in the skin of the thighs. In twelve minutes the operation was begun. Four minutes later the bone was sawn through without the patient feeling anything. Pulse as before. The manipulation of the limb not felt. Towards the end of the operation the pulse was, if anything, more regular and forcible than before the operation, and the patient was beginning to be aware of the movements of the stump, but they were painless.

The parts divided in the middle of the very voluminous thigh were all œdematous, and all the veins blocked with firm thrombi. The main artery gave very little blood, but was tied for safety's sake, as were also a few smaller vessels. As there was evidently septic matter in the soft parts, the wound was packed and only partially closed, being covered with a thick gauze dressing.

There was no shock at all, and the next day the sister of the ward said quite spontaneously that she was amazed at the condition in which the patient came back to the ward from the operating theatre: she seemed relieved, had a good colour, and was cheerful, offering a very striking contrast to most other patients returning after severe operations. There was no sickness, no headache, and no pain in the back. Dr. Bradford, who examined her later, found the condition of the heart and pulse much improved. The temperature fell at once to 99·6° F. from 103·2° F. The course of healing was that of a septic wound with oscillations of temperature. The ends of the flaps necrosed and came away in thin strips. It was evident that I might have removed the limb higher up in sounder tissues with advantage. The patient was very heavy and difficult to dress and a small bedsore formed. At the present moment the stump is healing nicely and the temperature is normal, everything else being also satisfactory.

This case is one which illustrates in an unusually striking manner the advantage of having at one's command for special cases the new method of producing analgesia by injecting the lumbar sac with an analgesic compound. Owing to the advanced heart disease, the dyspnœa and high fever, the administration of chloroform or ether would, in the opinion of all who saw the case, have been in the highest degree perilous. As one physician expressed it, it would have been almost certainly fatal. And yet the limb had to be removed high up with as little pain and shock as possible. I was glad therefore that I had had a considerable experience of spinal analgesia in other cases, and had mastered some of the details of the method, as well as studied it in its other aspects.

The conditions produced by the lumbar injection were for the operator almost ideal. All the terrible pain in the limb was gone within seven minutes and the muscles were relaxed, so that the thigh could be moved about during the operation without the patient being conscious of any movement. She was semi-recumbent on account of her cardiac dyspnœa but was prevented from seeing anything. She was quite cheerful and good-humoured all the time. While I was stitching up the wound I asked her if she had felt anything at all. She answered "nothing at all." I asked as to the sawing of the bone, and she answered that she heard the saw going and that she thought she felt slight movement, but that it was probably "imagination," but there was no trace of pain at any time. Another advantage of this method was that food and stimulants could be given after the operation as if nothing had happened.

There may be many opinions as to the place which spinal analgesia will take in surgery, but in this case it was beyond all doubt an unmixed blessing, and I believe has saved a life which under any other treatment would have been sacrificed. As to the details of the method I hope to have something to say later in a special paper.

May 7th, 1906.

ON CERTAIN DERANGEMENTS OF THE KNEE.

By ROBERT JONES, F.R.C.S.

GENTLEMEN,—To most practitioners the deranged knee is not a stranger, and I think many will admit to a sense of uneasiness when cross-examined as to definite diagnosis. There have been recent efforts to cavil at the vague import of the phrase "derangement," but as a general term it is sufficiently explicit. It conveys no suggestion of progressive disease, but rather of mechanical disability. As we shall soon see, there is often great difficulty in making an accurate diagnosis. A young lady waltzing, an athlete in a football scrimmage, an old lady removing the bedclothes with her foot, a miner working with fully-flexed knee, all feel something give. The events following this common symptom may widely differ. In one case the joint may have locked, in another acute pain is present, in another obstinate effusion occurs, in another neither pain, effusion, or locking, and, despite this, the diagnosis in each case may be the same. Accurate knowledge, therefore, is not quite simple,

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and the most experienced surgeon when he opens the knee is sometimes surprised at what he finds.

The derangements which we will discuss are synovial fringes, joint lipomata, loose bodies, exostoses, injuries to the crucial ligaments, recurring dislocation of the patella, injuries and displacements of the internal semilunar cartilage.

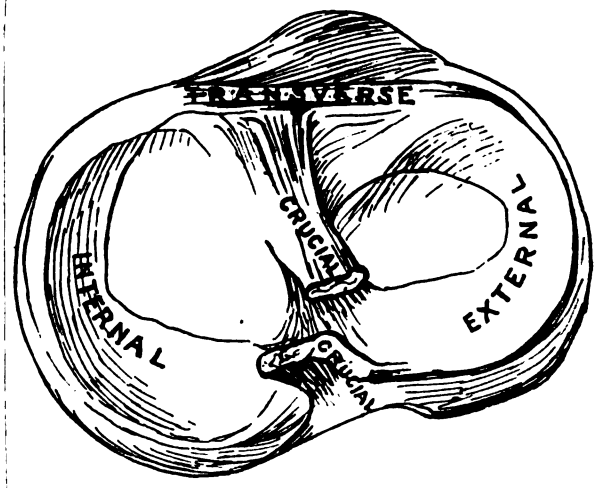
We will deal first with the most common of these affections—injuries to the semilunars—and to simplify the subject, you will pardon me if I take you over certain anatomical points in reference to the mechanism of the knee and the attachment of the cartilages.

Anatomy.—The knee is a hinged joint where the bones, apart from soft structures, are not adapted to one another. Its strength depends upon the tendons and ligaments which surround it and upon two principal ligaments, the crucial, which lie within. How strong these ligaments must be is evident when you consider the shape of the knee-joint, no part of the bony conformation in any way calculated to resist this displacement, and yet the soft structures are sufficiently effective to resist dislocation in spite of the leverage of the two longest bones in the body. Of the capsule I need only say that it acquires most of its strength from bands derived from surrounding tendons. The motions of the joint are antero-postero, gliding and rotation of tibia upon femur. The ligaments allow of no other movement. In no position of the knee is lateral movement allowed, but when flexion is extreme considerable rotatory movement can take place, and every time the knee is fully extended a certain degree of outward rotation normally occurs. The ligaments which prevent side to side movement during extension and during slight flexion are the laterals and during the more acute flexions the crucials. It is obvious, however, that in conditions of strain the muscles are valued accessories—the rectus in front and the hamstrings behind—but they are quite inefficient as substitutes for either the crucials or the lateral ligaments should they be put out of action.

Mr. Joseph Griffiths, of Cambridge, who has written a thoughtful article on the mechanism of the knee, having severed the internal lateral ligament, found that during extension of the limb the tibia could be appreciably bent outwards and the articular surfaces of tibia and femur be correspondingly separated. If the same joint were flexed the crucials prevented such movement. In a case of complete rupture of the internal lateral ligament in a wrestler I confirmed this experiment clinically, and in another patient, where a portion of bone accompanied the separation of the ligament, no lateral movement was permitted during acute flexion. A strain upon the ligament is much more severe when it is twisted as well as stretched. Now, how can such a twist be applied? It takes place when the knee is flexed, the foot abducted, and the femur rotated inwards. It is, in fact, in this position that strain and rupture of the internal lateral ligament and displacement of the semilunar cartilage almost always occurs. Now I would desire to point out that there is a very close association between injuries to the lateral ligaments and displacement of the semilunars, as we shall see more clearly after glancing at the anatomy of the semilunars.

The internal and external semilunars (Fig 1) are two dense crescentic plates of a compact structure

FIG. 1.



attached by their outer convex margin to the capsule of the joint. They cover almost two thirds of the articular surfaces of the tibia. On section they are wedge-shaped, tapering from their outer thick base to their inner free border. They thus increase the concavity with which their femoral condyles articulate. Their lower tibial surfaces are flat, and both the tibial and the femoral surfaces are free, and what is important clinically, both surfaces are covered with synovial membrane; so that in all movements of the knee-joint the femur and tibia put direct pressure upon the reflecting synovial sac.

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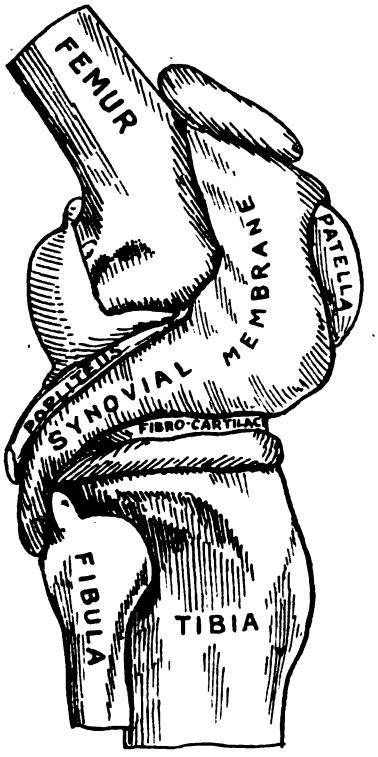
This bears a close relationship to the effusions which so often follow injury. Both cartilages taper to their attached ends, which are in front of and behind the tibial spines. The attachments of the external cartilages are within those of the internal. For this reason the external cartilage is more circular than the internal, which is oval in shape, and which is longer from before backwards. An important point to remember is that from the closer approximation of its ends the external cartilage is more movable than the internal, and this is made more pronounced by the laxity of the part of the capsule to which it is attached. In thin people the prominent inner margin of the internal cartilage can be felt between the bones in front of the internal lateral ligament (Fig. 1). The rim of the external cartilage in front lies more within the joint, and cannot be felt (Fig. 2). Both cartilages

Fig. 2.



in their convex border are attached to the capsule of the joint; both cartilages have attachments to the posterior crucial ligaments behind (Fig. 1). The external cartilage is not attached to the external lateral ligament, while—and this, as we shall see, is very important clinically—the internal cartilage has a very firm attachment to the internal lateral ligament. The union of the external cartilage to the capsule is, as I said, less firm than in the case of the internal, because the anterior end is nearer the centre of the joint and posteriorly the tendon of the popliteus separates it from the capsule (Fig. 2). The coronary ligament is a name given to that part of the capsule between the attachment of the semilunar above and the tibia below.

As we have seen, bending inwards of the knee in the extended and semi-extended position is prevented by the internal lateral ligament. Bending inwards of the knee during the fully flexed position is prevented by the crucial ligaments. These two anatomical facts should be remembered

Fig. 3.

in their relation to displacement of the internal semilunar. One can thus easily understand how it is that the internal lateral ligament so frequently suffers injury. It is subjected to strain whenever the foot is firmly planted and abducted and the knee either slightly bent or kept extended (Fig. 3). If, in addition to this, as in the stroke to leg in cricket, or the final act of the bowler, body weight with rotation of the femur is added, we have produced that stretching and strain which Griffiths has proved to be so harmful to the integrity of the ligament. Indeed, the strength of the knee-joint can be well gauged by the resisting power of the internal lateral ligament.

The internal semilunar is attached to the tibia in front by fibrous bands, running towards the

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tubercle. Circumferentially it is attached to the tibia by structures—and this is an important point—which allow the cartilage to be lifted fully a quarter of an inch. In the middle, however, it is braced up by a firm fibrous band which becomes merged into the strong internal lateral ligament. The point of practical interest in connection with this attachment is that this ligament keeps the cartilage fixed to the femur in rotation of the joint. Furthermore, any traction exerted upon the upper part of the ligament falls upon the so often displaced anterior portion of the cartilage. If the internal lateral ligament be torn above the level of the joint, the cartilage remains upon the tibia; if torn below the level of the joint, it follows the rotary movement of the femur. If the ligament be ruptured above and below the level of the cartilage, it becomes a very simple matter to push it within the joint, a not unusual position to find it in. It would appear, therefore (Griffiths), that displacement or fracture of the cartilage can only take place when its circumferential and suspensory attachments are torn, and when the internal lateral ligament is stretched or ruptured. This allows a separation of the joint surfaces and a slipping inwards of the cartilage, as the separate bones snap back, the cartilage is nipped, and the knee is fixed in a very painful manner. The pain and effusion can be ascribed to injury of the synovial membrane, with which, as I have already stated, the cartilages are covered.

I have very little doubt in my own mind that in the great majority of instances the cartilage is displaced inwards, and in those instances where a protrusion can be felt from the outside it is due to a buckling of the cartilage, as shown in Fig. 4. The arguments in favour of displacement inwards are many. Although I have personally operated upon over one hundred cases, I have never yet met with a displacement outwards not accompanied by a correlated displacement inwards. A displacement outwards could not possibly produce a locking of the joint; this can only occur when the displaced cartilage, acting as a foreign body, gets jammed between the bones. It is this internal displacement which gives rise to the acuteness and painfulness of the attack. We must all have been struck with the absence of anything abnormal from an external examination of a knee subject to recurrent attack.

Now, what are the symptoms of displacement of the semilunars? The most constant symptom is a sudden inability to fully extend the joint; the cause is strain thrown on the internal lateral ligament while the knee is flexed and the femur rotated inwards (Fig. 3). The force necessary in a first injury is generally great, the pain acute, and the victim, if an athlete, hobbles laboriously with flexed knee off the field. He is afforded a certain degree of relief by his friends in the pavilion, who fully flex his limb and then extend. When he reaches home the practitioner finds a distended knee, painful to manipulate. The strain which is most painful is that employed in stretching the structures on the inner side of the knee, and this is most acutely felt over the internal lateral ligament. In a certain proportion of cases, even with the knee fully extended, a gap between the bones on the inner side can be obtained by manipulation. For several succeeding days there will be pain on pressure, if exercised over the articular edge of the tibia opposite the ligament, and, generally speaking, some pain between the bones on pressure to the inner side of the patella. In a fortnight or three weeks, after rest and massage, the patient is by common consent allowed liberty to walk. As we shall see later, this liberty is a mistake and the outcome of very indifferent surgery. Days, weeks, or months, may elapse when again a train of symptoms, similar in character, but generally milder in effect, occurs. Many successive attacks, some grave and followed by effusion, some so trivial as to merely incommode, mark retrogression. In nearly all cases there is a history of strain or injury, the initial displacement as a rule being the most severe, while in the constantly recurring case any eversion of the foot may give rise to the displacement. I have had occasion to operate but four times for an external semilunar, which in my personal work only represents a 4 per cent. proportion. The reason the internal cartilage is so frequently involved is dependent entirely on anatomical grounds. Being, as we have seen, more firmly fixed by ligamentous attachment than the internal, there is not the give and take about it which you find in the less controlled external cartilage, especially in the rotary movements of the joint. Again, in the normal relation of femur to tibia the line of force is carried through the inner side of the knee; this is further emphasised by the abducted position of the foot,

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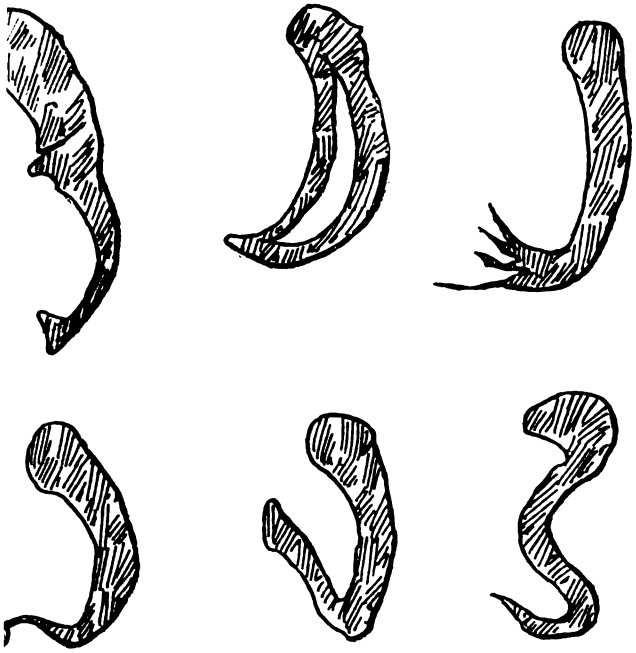
which, if exaggerated, produces outward rotation of the leg. Another reason may be found in the fact that external rotation, which is a direct strain on the internal semilunar, is always more pronounced than is that of external rotation.

What is the character of injuries which we find to the semilunars? Of twenty-six cases upon which I operated last year, thirteen were torn from their anterior attachment; four were longitudinally split; two were displacements of the posterior horns; two were loosely bound circumferentially; two were attached by their cornuæ and detached into their joint centrally; three, which presented interesting symptoms, exhibited altered anterior portions which were thickened like peas attached by a pedicle to the inner side of the spine (Fig. 4). In my whole

FIG. 4.



series I have only come across six cases of complete fracture of the semilunars, and in each case the fracture has been opposite the internal lateral ligament. Some cartilages which I have removed have undergone changes which render them barely recognisable, some nodular and fibrous, others so thin and frayed that they tore when grasped by the forceps, and had to be removed piecemeal. Before discussing the differential diagnosis and supplying illustrative cases, I should like to refer to certain other conditions which give rise to symptoms not dissimilar to those of injured semilunars. First amongst these allied conditions I would place loose bodies. They may be cartilaginous, fibrous, or fatty. When fibrous they usually result from unabsorbed blood-clot. The lipomata may be small or large, and develop in the synovial tissue, being attached usually by a pedicle. The larger lipomata are usually found to the side of the ligamentum patellæ (Fig. 5). Hypertrophy of the synovial villi is frequently confused with a damaged semilunar. The condition is much more common than is usually suspected, and I have frequently met with it when exploring joints. It is usually primarily the result of synovitis, frequently later the cause of it. The whole joint may become papillomatous, which does not interest us at present,

FIG. 5.

or a few isolated fringes may be found generally where the synovial membrane meets the cartilage. They are often pedunculated, may be found anywhere along the articular margin, and are composed of fibrous tissue, fat, and blood-vessels.

On one occasion, failing to find any injury to the semilunar in a case with typical symptoms, I enlarged my incision and found a detached ligamentum alaria (Fig. 6) lying in the intercondyloid notch. Its removal resulted in a perfect recovery.

I should like to make an effort, imperfect though it needs must be, to differentiate between these various arrangements.

Displacement of the internal semilunar cartilage is diagnosed by the acuteness of its onset, by the yielding of and pain on pressure over the internal

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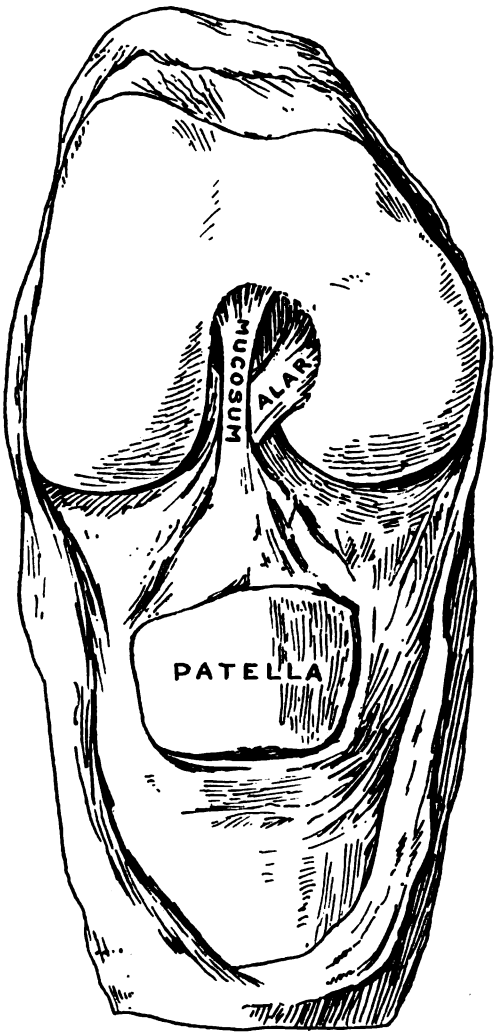
lateral ligament, with tenderness, not so pronounced over the inner side of the patella, and particularly by a locking of the joint.

Displacement of the external semilunar cartilage produces similar symptoms, the pain and yielding being referred to the outer side.

Loose bodies can usually be found and isolated

Fig. 6.



by the patient. They often lock the knee, but as a rule very transitorily. The symptoms are generally sharp but not acute; effusions often occur, and the body or bodies may be felt at different times in different places, because of their nomadic habit.

The nipping of a synovial fringe is less acute in its primary occurrence than is that of a displaced cartilage; the pain is strictly local and is not participated in by the internal lateral ligament. Frequently a prominence may be found over the site of pain, and no matter how often the nipping occurs, effusion follows. Creaking in the joint is a frequent accompaniment, and the nipped fringe may often be felt.

The symptoms of lipomata sometimes include locking of the joint. There is swelling without much effusion. The enlargement is usually each side of the patella tendon. The symptoms are never acute, and only very rarely is there pain on pressure.

Rupture of the crucial ligaments, of which I shall relate a case, is the accompaniment of so severe an injury that other structures participate in the general strain. The tibia can be made to glide in a to and fro direction, and when the knee is flexed lateral movements are free. If the lateral ligaments are torn lateral movements in the extended positions are also free. A case has been reported by Mayo Robson, verified by operation, and another, not verified, by Stanley Boyd. I have operated upon one such case.

I am quite conscious that despite any amount of care in diagnosis, error is inevitable in a number of cases, and only exploration can accurately inform. For some weird reason on occasions all the symptoms may point to injury on the inner side and when an exploration is made no lesion can be ound. One such case I will relate.

A young man of firm muscular development received an injury during a football scrimmage. His knee locked and he was laid up in bed for three weeks. He complained of pain, as far as he could recollect, both on the inside and on the outside of the patella. In five weeks, when jumping over a ditch his knee locked, but only very transitorily and he was able to reduce it. He complained of pain on the inner side of the joint over the lateral ligament. On three subsequent occasions something gave on the inner side of the knee, and when he consulted me, some weeks after his last attack, all tenderness had gone. I decided that the internal cartilage required removal, but when I explored it I quite failed to find any defect in its appearance or behaviour. I examined for loose bodies or hypertrophied fringes, but quite without success. Thinking that perhaps a loose body might be found on the outer side, I explored

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that aspect of the articulation through a second incision. To my great astonishment, instead of a loose body, I found that the posterior horn of the external cartilage was doubled over and displaced forwards. Complete removal of this cartilage resulted in recovery.

I will relate another case to show how a displaced cartilage may not give rise to any typical symptoms. I saw a boy of eighteen who periodically had effusions in his knee, accompanied by much creaking but no pain. Three years previously he fell, but gave no history of locking, and walked a mile home. Next day his knee was much swollen and for a fortnight he rested it. For three years this effusion, with short intervals, continued. The knee felt stiff; it sometimes pained when fully extended, but never once did he feel anything give in the joint, nor was pain ever referred to in any localised spot. Every variety of treatment had been tried and discarded before I saw him—rest, splints, massage, exercises, electric baths—but all of no avail. I determined to evacuate the joint by lateral incisions, and if necessary inject the cavity with iodine and alcohol. On entering the articulation and peering around, I saw the anterior end of the internal cartilage, thinned and detached, and lying very flat to the inner side of the anterior crucial ligament. I removed the cartilage and evacuated the contents of the joint. Movement and massage were started in a fortnight, and the patient made a slow but uninterrupted recovery. I mention these two cases as being of considerable clinical interest in order to emphasise difficulties of accurate diagnosis. You must not think, however, because of these very exceptional cases, that it is useless to attempt classification. On the contrary, the exceptional case is necessarily that where a diagnosis is rendered difficult or impossible.

The most common injury to the knee-joint is a sprain or rupture of the internal lateral ligament. As we have seen, this should not be mistaken for a slipped cartilage. In the one case a derangement is experienced, in the other it is never so. Next to the injury of the internal lateral, which is often commonly called a sprained knee, injury to the inner cartilage is most frequently met, and the problem of treatment must be solved. I maintain that a great distinction should be made between a primary and a recurrent luxation. I never operate in any but recurrent cases, and I have no doubt that were the first offence intelligently dealt with and at once, the proportions of such recurrence would be materially diminished. This may not be quite orthodox, but I cannot help feeling that it is quite true. Surgeons point to the thinned and battered cartilage, fractured here, displaced there, and ask, "What can mechanism do here?" It is quite true! A time comes when operative measures are inevitable, but that is only when cartilages inefficiently reduced, or far too frequently nipped, show the evidences of their maltreatment. The first displacement will probably not be accompanied by any structural change, and the knee will very often quite recover, if appropriately approached. (*a*) Reduction must be absolute. (*b*) All movement of the cartilage must be checked until union of the torn structures is complete. (*c*) No lateral strain must be allowed until the torn or stretched internal lateral ligament has recovered its tone and strength.

With regard to reduction it does not matter what method is employed, provided easy full extension is secured. Too often, more especially in badly displaced or protracted locking, the surgeon is content to acutely flex and extend the joint, and if at last, though full extension is not quite free, a back splint is applied, and extension, which should be secured by manipulation, is brought about by pad and bandage, what probably happens is this: Reduction not being quite complete, there remains a slight obstruction to full extension, and the pad and bandage tightly compress the cartilage between the bones. Let us remember as we would an axiom, that we can only be assured of reduction when the knee can voluntarily be held in complete extension. I learnt this lesson some years ago. A youth subject several times before to slipping in his knee came to me straight to the pavilion from the cricket field. His joint was locked at an angle of 40°. I put the limb through the orthodox manipulation, and it came nearly straight. I tried again, with a similar result, and felt inclined to be content. "It is not in," he said, and, humiliated, I strove in public for five minutes, when without any objective sign he cried, "Now I am right," and the knee could forthwith be held extended. In reading text-books one would conclude that reduction of a displaced semilunar is the easiest possible routine. This is

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a deduction based on very limited experience, for some cases are especially difficult to fix. In the majority of instances reduction is easily effected; in a few recent cases it is difficult; in some old cases it is extremely troublesome. What is the best routine manipulation? Acute flexion, lateral deviation and rotation inwards, and full extension. Acute flexion is always painful, but it is the only position where internal rotation of the joint is most free; lateral deviation separates the bones which hold the cartilage, and full extension allows of readjustment and places the limb in such a position as to permit of accurate union of the internal lateral ligament.

Retention of the cartilage in a fixed position.—This can only be secured when the limb is fully extended. In all rotary and lateral movements of the joint the cartilages participate. It follows, therefore, that both these movements should be prevented if we are to strive for accurate union of the torn attachment to the cartilage. Rest of the limb in a fully extended position is therefore indicated, and rest should be accomplished in bed so long as effusion lasts. Do not let us forget that a joint distended with fluid relaxes by elongation all the protective soft structures of the joint, and the more rapidly we attain absorption the more we avoid a prolonged weakness. When all fluid is absorbed, and the knee resumes its normal aspect, we proceed to the next stage. It may be days, it may be weeks; if slow, massage may help, but under no conditions must flexion be allowed. Flexion, as we have seen from the anatomical conditions, interferes with the moorings of the cartilage and the integrity of the lateral ligaments, both disturbances quite inimical to union of the torn structures. The third indication is to prevent for a prolonged period lateral deviation of the joint. We have seen that in derangements of the cartilage strain or rupture of the internal lateral ligament is a necessary incident; it follows, therefore, that during recovery no lateral strain should be thrown upon the knee.

Now, you will remember that the abducted foot, the flexed knee, and the lateral strain in combination are the direct causes of the displaced semilunars. Obviously, therefore, our object is to prevent abduction, lateral deviation, and flexion.

One of the simplest methods of preventing strain upon the internal lateral ligament is to walk upon an inverted foot with inturned toes. Seeing, as I do, a great number of cases of rickets, I am in the habit when treating a genu valgum in an early stage of insisting that the patient should walk with parallel feet so aided by altered boots that the body weight is deviated from the inside to the outside of the tarsus. This throws a slight strain upon the external lateral ligament, and enforces pressure upon the inner articular surfaces of tibia and femur. So effective is this pressure that it generally suffices in a few months to correct the deformity. You can understand, therefore, what an important ally we have in so simple a measure to keep from strain the injured internal lateral ligament. In cases where the cartilage displaces at frequent intervals I order an alteration in the heel and sole of the boots. The heel is elongated and raised on its inner side, and the inner side of the sole is fortified by a small wedge of leather. When the patient stands in such a boot the foot is inverted, and the lateral strain which is needed to displace the cartilage is avoided. If, in addition to this, the young athlete be told to walk with an inturned toe, and to run pigeon-toed, strain is inevitably thrown upon the external lateral ligament, while the internal lateral ligament is protected. Although immunity from displacement cannot be assured by this device, it materially lessens the occasions and becomes a valuable asset amongst our remedies. In addition a splint must be employed. I have brought with me two kinds, either of which you will find of great help. The first I show you is devised by Mr. Griffiths, and made by Offord, of Cambridge (Figs. 7, 8). As you see, its construction is very simple. Two flat bars on each side are fixed together by a free hinge, which lies at the middle of the back of the knee. These bands are so placed as to strenuously resist any lateral pressure, and to a large extent, if not wholly, rotation. I frequently use the cage which you see made by Critchley, of Liverpool. These are lateral supports, as you see, and, in addition, a pad is employed which places firm pressure upon the anterior portion of the cartilage when forced outwards in compensation to the middle part of the cartilage, which is forced inwards. In all recurrent cases which do not need operation, and in all cases of first displacement sufficiently recovered for flexion to be allowed, one orders a splint, alters the boot for the purpose of inverting the foot, and disallows walking except with

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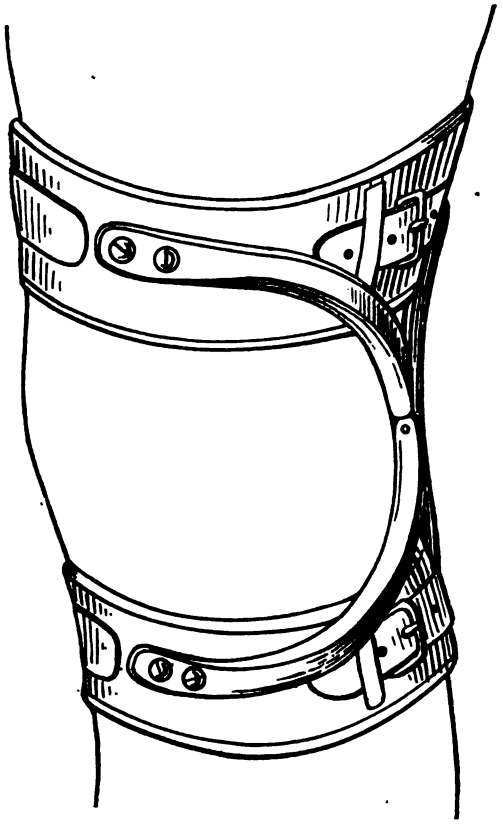
parallel feet and running except with inturned toes. In addition to this, the quadriceps, as the guardian muscle of the knee, must be energetically massaged and otherwise kept fit and strong. May I once again emphasise the importance of adequately treating the first displacement, and of impressing upon you that if the treatment is conducted on the lines I mention quite a large proportion of cases will make excellent and permanent recoveries. The recovery depends upon complete reduction and uninterrupted union and consolidation of both

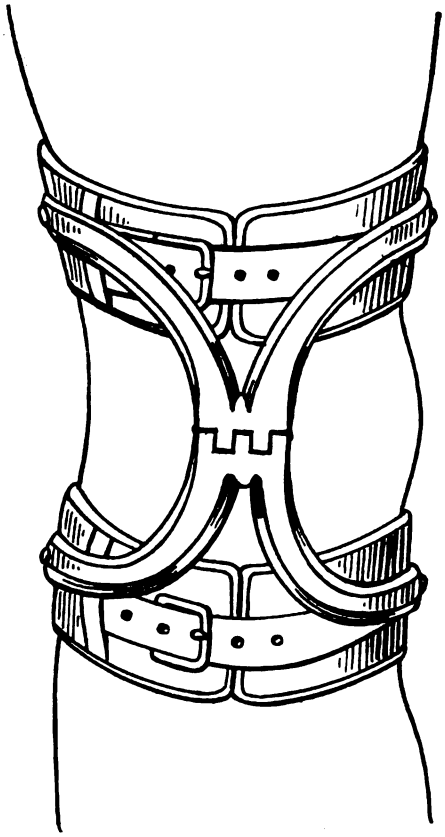
FIG. 7.



the internal lateral ligament and the ligamentous moorings of the cartilage. In operating, as we so often do, on old injured cartilages, we are apt to be unduly biassed when we see their thinned and battered edges. This condition can only be the result of frequent injury, and should not discourage us in the mechanical management of a first slip. I have at the present time many athletes playing tennis, football, and cricket who have undergone a rigid mechanical treatment for undoubted displacement. The question will be naturally asked, "What are the indications for operation?" I will try and define my attitude in regard to mechanical and operative treatment.

In the first place *I refuse* to operate in any case I see early, the subject of a first derangement. *I discourage* operation in those recurrent cases when the symptoms are transient and not followed by irritation of the joint. *I strongly urge* operation in those cases where a recurrent displacement is at times followed by acute symptoms. I advise it in all recurrent cases where a strenuous athletic life

FIG. 8.



is a means of livelihood or a physical necessity. I think operation absolutely imperative in the case of men who work or stand in dangerous places and where a yielding of the knee may lead to serious consequences. Practically age has no influence over me if the indications I mention are present. I have operated upon a boy of fifteen and a woman of fifty-four.

Is there any danger apart from periodic trouble in a slipping semilunar? I can clinically support Mr. Arbuthnot Lane when he says it may prove

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the exciting cause of tubercle. Frequently I have examined patients with tubercular knees and patients with rheumatoid arthritis whose early history was associated with internal derangements of the knees, and in several cases of badly recurring injuries I have found sometimes congestion and sometimes erosion of the bone and cartilage upon which they rested. One case I will refer to which bears upon the point. Some eight years ago a youth, æt. 19 years, consulted me concerning a cartilage which two years previously became detached. Several recurrences took place, some of them followed by synovitis and rest in bed. I urged removal of the cartilage. The youth, however, peristently refused my overtures and only grudgingly and spasmodically wore the splint. In less than eighteen months I noted that one of the attacks left him with a thickened and sensitive knee, which in time refused gradually to fully extend. At the present time he has recovered from a typical tubercular knee, firmly ankylosed. This case is specially interesting to me as I have been able to watch the development from the beginning. Another case, a man æt. 25 years, a boiler-maker, sprained his knee. Later he became a frequent patient, with slipping internal semilunar. Three years after creaking and swelling of the joint led to the suspicion of what Goldthwaite terms the atrophic variety of rheumatoid arthritis, which radiography fully confirmed. Of two cases, carefully watched, each refusing operation, one resulted in rheumatoid and the other in tubercular change. I would suggest, therefore, that this danger should be kept well in view, and that patients with either a tubercular or rheumatoid diathesis subject to recurring derangement should early be persuaded to have the exciting cause removed.

Is operative treatment invariably successful? The answer is emphatically, No. In the great majority of cases a perfect recovery may be predicted; in a certain small percentage of cases the symptoms recur. The recurrences were far more numerous some few years back, when the cartilages were sewn to their tibial attachments. The same causes which accounted for the initial displacements produced the recurrence. Since it is customary to exsect the cartilage other causes than a displacement must be searched for in answer to symptoms. It will then be discovered that the so-called recurrence is due to an overlooked accessory factor in the production of the symptoms of derangement. I will illustrate this by very briefly referring to cases.

A young woman of 24 complained of a locking which was referred to the outside of her joint. It had recurred on several occasions, was sometimes accompanied by pain and sometimes followed by effusion. On occasions the slipping was not accompanied by locking. On opening her knee-joint the external semilunar was found torn anteriorly and I removed the anterior half. A month did not elapse before slipping again occurred, and on careful inquiry as to locality no reliable history could be obtained. I therefore opened the knee from the inner aspect, as I felt I should have seen any abnormality had it been present on the other side at my first operation. Floating by a thin pedicle a small fibrous nodule presented, which was removed with scissors, the knee recovering its normal function.

A miner of about 30 complained of a slipping in his joint, sometimes accompanied by locking and at other times not. There was always sensitiveness over the articular border, between the patella and the internal lateral ligament. There was often effusion. On opening the joint I found the anterior portion of the semilunar abnormally free, but apparently not detached. Having several times found a similar condition, for which I exsected the cartilage with restoration of normal function, I exsected here, and for some months no symptoms were experienced. Later, however, quite similar symptoms were complained of, and pain, again referred to the inner side of the knee. I followed the line of the old scar, and searched for a cause. I was almost closing the wound, when internal rotation of the tibia dislodged a loose body which shot out of my wound. Both these cases illustrate the danger that more than one mechanical defect may exist in a joint, and the removal of merely a part of them will not cure the patient. We, therefore, must carefully inspect one joint when it is opened to make sure that we have attacked all those elements likely to give trouble.

A very common cause of derangement of the knee is the presence of so-called loose bodies, or what the Germans call "joint mice." They are sometimes difficult to diagnose from injured semilunars. The attack is often an extremely painful one. The patient feels sick and faint, and often

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falls in agony. The pain is usually of brief duration, but synovitis frequently results. Locking is uncommon but sometimes does occur, but is immediately, though painfully, overcome. They may result from detached degenerated processes of cartilage following injury, from displaced synovial tufts as the residue of blood-clot, or as broken off osteophytes in commencing arthritis.

These are only a few of their modes of origin. They may be as small as a millet-seed or as large as a fig. They may be any shape and exist in any number. Often they are found facetted; they may be fairly soft or extremely hard. Their structure is often cartilaginous, generally without corpuscles, more rarely of bone, and when bony usually do not present Haversian canals. The surgeon can often detect these bodies by careful manipulation, now in one part of the joint, now in another. They are as slippery as eels, and usually the patient is himself best able to fix one in a temporarily immovable position. One can only be sure of the diagnosis when the body can be felt and its nomadic character assured. If a knee-cap fails to keep the bodies from between the bones, operation must be the treatment.

A condition very puzzling, but one which most of you have probably encountered, is a loud click on acute flexion and sometimes on complete extension. This condition has been described as "trigger knee," because of its characteristic snap. It is most frequently experienced when extension is all but complete. You place your hand upon the patient's knee and ask him to extend his joint; this he does almost perfectly until about 170°, when the remaining distance is performed suddenly accompanied by a click and usually an outward rotation of the tibia. The cause is difficult to explain and is generally ascribed to a disturbance of the movement of the external semilunar, which is successively caught and freed between the joints. Two cases I operated upon may throw some light upon what the surgeon may expect to find.

A bank clerk not given to athletic sports presented such a knee. He complained of a sense of insecurity, more, he said, imaginary than real, and he told me that his health was undermined because of the nervous irritation it caused. The click was not accompanied by pain and never by effusion. He refused the suggestion of mechanical treatment, and I explored his joint. I was desirous of not opening his joint by an interior flap, and without any real reason I entered the joint from the inner instead of by the outer side. At once the cause was apparent. Tapering from the anterior portion of the cartilage and attached by a cartilaginous pedicle was a chondrous structure the size and shape of a flattened pea. It lay loose just on the inner side of the anterior crucial ligament, not so far back as the spine. On extending the joint the click was heard, but I could not, of course, see exactly what occurred. I removed the anterior portion of the cartilage, the tadpole ending, and I was able at once to fully extend the knee without the occurrence of the sound. There was a distinct wearing of the cartilage over the short track traversed by this nodule. The patient made a complete recovery. My second case very closely resembled the first. Clicking on

FIG. 9.

extension of the joint was constant but not complained of. Periodically, however, the joint locked, and it was for this reason I was consulted. The locking was on the inner side, and I therefore approached the joint from this aspect. The anterior portion of the cartilage was thin and cordlike, quite detached, and ended in a thickened extremity (Fig. 9). On removal of the cartilage the clicking disappeared, and the patient also recovered the proper function of his joint. I shall be very interested in future cases where this puzzling symptom is present. The mechanical treatment of such a condition consists in the application of a splint which, while allowing movement of the knee, prevents its full extension.

Some years ago I operated on two cases where a clicking could be felt at the back of the knee. In both instances it was due to the slipping of the semi-membranosis tendon over a small tibial

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exostosis. In both cases the disability was frequent but not constant, and at times weakness and soreness were present. A third case was both interesting and instructive. A man, æt. 24 years, complained periodically of a locking knee. The locking was accompanied by scarcely any pain, it seemed to take place in the popliteal space, never in front of the joint. Generally there was no great difficulty in releasing the leg; the patient by very rapidly flexing the limb at once freed it. On occasions, however, the locking was very complete, and the knee would remain flexed for some time until medical aid arrived. An exostosis could be felt just above the insertion of the biceps tendon, which when removed was bicornual, like the letter Y and the tendon sometimes slipped over the whole of it and at others was received between the horns. In each of these cases removal of the exostosis remedied the defect.

Rupture of one or both the crucial ligaments is a rare and serious accident, and would not be included amongst the derangements were it not for the uncanny symptoms to which it gives rise, involving as it does the joint in abnormal movement.

In 1902 Mayo Robson reported the case of a miner who met with a severe accident, being buried in earth, and sustaining, amongst other injuries, a fracture of the leg and of ribs. After many weeks in a country hospital he was admitted into the Leeds Infirmary with a swollen knee. When the muscles were braced up the bones were in good position, but as soon as the muscles were relaxed the tibia fell back until stopped by the ligament of the patella. By manipulation the head of the tibia could be brought forward in front of the femur, and there was also free lateral movement. It was decided that not only were the ligaments generally relaxed, but that the crucials had been ruptured. When operated upon both crucials were found completely torn from their upper attachments, the ends being somewhat shreddy. They were stitched in position by means of catgut ligatures, and some years afterwards when examined the joint had almost wholly recovered its functions. Although I have seen several cases where the diagnosis of rupture of the crucials, either anterior or posterior, was suggested, I have only had an opportunity of operating upon one case; it was that of a barman who fell down some cellar steps and dislocated his knee and for three weeks was kept at rest. He was then allowed to move about the house, and consulted me some ten weeks later. He had some pain in the knee, a little effusion, and, as he described it, "no dependence" on the joint. It slipped about, creaked, and was only moderately useful, and that only when a splint was worn. On examining the knee, the leg could be rotated inwards, but not outwards. The patient complained of pain whenever the knee was hyperextended, this being to a very limited extent possible. The tibia could be slightly displaced forwards in extension. I opened the joint by a free anterior incision, dividing the patella ligament, and found a complete rupture of the anterior crucial and considerable reddening of the synovial membrane. I freshened the ends, which were very frayed, and as they could not be brought together I plaited some chromicised gut as in nerve-union. For six weeks the patient was kept in bed, and in eight months after the operation the stability of the articulation was secured. In several cases I have noted a weird lateral and antero-postero mobility, with varying functional disturbances, but only in one case have I had an opportunity of exploring the knee. In the early part of last year I examined a boy of fifteen who fell in scaling railings and was caught by the foot and held head downwards. I detected a separation of a small piece of bone from the inner femoral condyle at the attachment of the internal lateral ligament, and there was free mobility in all directions. It was obvious that in addition to other lesions both the crucials were ruptured. Immobility of the joint for two months and subsequent control of the lateral ligaments resulted in a good recovery. We may, I think, conclude that rupture of the crucials is not an uncommon incident in dislocations of the tibia and other grave lesions, and that it is amenable to mechanical treatment if such treatment be sufficiently protracted. In old, neglected cases the gliding tibia is the characteristic derangement. If the anterior crucial alone be ruptured or elongated, the leg can be displaced anteriorly, it may be hyper-extended, and it can be rotated inwards.

If the posterior or stronger ligament be similarly injured the tibia can be displaced posteriorly. If both are ruptured the tibia can be moved backwards and forwards, and if the knee be flexed outward rotation of the tibia is increased.

Slipping knee-cap.—There is another derangement of the knee which presents characteristic symptoms. I allude to a slipping patella, or what more correctly should be termed a recurring dislocation of the patella. The displacement is nearly always to the outer side. The etiology of the displacement is suggested by the anatomical fact that the line of action of the quadriceps in the axis of the thigh differs from that of the patella ligament in the axis of the leg (Fig. 10). When the leg is extended by contraction of the quadriceps the patella lying at the angle of the meeting of these

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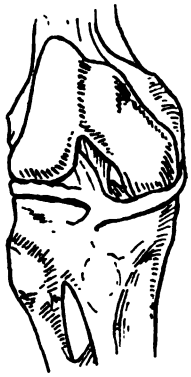
two axes must be pulled outwards as the muscle and ligament attempt to form a sharp line. Indeed, there can be little doubt that displacement would be very frequent were it not for a second anatomical fact. If you look (Fig. 11), you will find that the outer margin of the trochlear surface of the femur is so placed as to offer resistance to the outer deviation of the knee-cap. In a series of cases published by Goldthwaite he drew attention to what I have frequently observed, that the tibial tubercle is displaced too much to the outer side,

FIG. 10.

and in this way there is an increase in the angle of axis of the leg and thigh. If we alternately extend and relax the quadriceps, this lateral movement of the patella becomes obvious, and we at once note the manner in which the trochlear surface of the femur checks displacement outward of the patella. As Goldthwaite well says, "If for any reason the line of pull becomes less direct or the articular ridge less perfectly formed; if the capsule be weakened by the distension following some acute injury; if the patella tendon be abnormally long, so that the patella is drawn above the outer edge of the trochlear surface of the femur; or if the joint can be hyperextended so that during the muscular pull the patella is lifted away from the femur—in any one of these conditions the stability of the joint so far as the patella is concerned must be materially lessened."

The symptoms of slipping patella are obvious and fairly constant. The patient, perhaps rising from a chair, feels an excruciating pain in the knee and often falls. He is unable to move. The patella is found usually on the outside of the joint; effusion of fluid follows. In the recurrent case, if of long duration, no pain accompanies the displacement and no fluid is secreted. The reduction is easily accomplished. The leg should be first extended, the inner edge of the patella be elevated and passed back to its place. Patients nearly always complain of weakness of the knees, and operative measures are sometimes needed to correct the disability. I have transplanted the tubercle of the tibia into such a position as to

FIG. 11.



make the axis of the ligament and quadriceps one. In genu valgum accompanied by displacement of the patella I have performed a femoral osteotomy and maintained a strong extension in a small child. I have cut down upon the inner side of the capsule and seized it above and below by a pair of Kocker forceps. The forceps are simultaneously twisted and a longitudinal strip of capsule is plicated which draws the patella inwards; this plication is freshened and stitched and the joint is not opened. In one obstinate case complicated by genu valgum I divided the femur above the joint, transplanted the tibial tubercle to the inner side, and plicated the capsule. The best operation, however, in the uncomplicated is that devised by Goldthwaite, who splits the patella tendon, and after detaching the outer half passes it behind the remaining portion and implants it to the inside of the patella tendon. The tendon is stitched to periosteum and to the expansion of the tendon of the sartorious muscle (Fig. 12).

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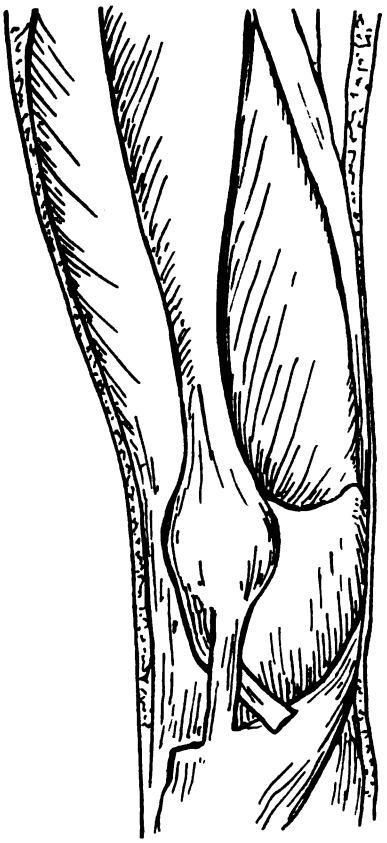
On excision of the semilunar cartilage.—There can be no greater tragedy in surgery than to infect a knee-joint for the sake of a derangement. I experienced one such tragedy, and the memory of it will always cling. Although obviously the greatest precautions should be taken, whatever operation be performed, on any part of the body, still, the slightest error in technique in removing irritant bodies from the knee will almost surely end in disaster. The knee-joint leaves no margin for mistake; in this respect it is not like the abdomen. Whoever undertakes to operate should be

FIG. 12.



scrupulously clean; he should pay, not homage merely, to asepticism, but allegiance. I prefer to have the knee cleaned with soap and water night and morning for a full week, and compresses of sterile water covered with mackintosh applied. On the evening before operation the knee is washed with biniodide of mercury and alcohol 1 in 1000, dried with sterile wool, and a dry dressing is applied. On the operating table in the morning I conduct a final scrub with mercury and alcohol 1 in 500, quickly followed by a rinsing with sterile water. The nurse, if she has to do with the sterilisation of the knee, should be herself free from infected hands. She should not have touched a doubtful case for days. Both when the knee is washed and at a later stage, when chemically sterilised, the nurse must wear indiarubber gloves, boiled for each occasion; less than this is not enough. The surgeon's hand should be thoroughly prepared, first in running water and then with spirit lotions. Ten minutes at least should be spent in the rougher cleansing before a germicide is used. When the hands are as clean as water and lotion can make them the antiseptic conscience will still distrust, and the surgeon must make assurance doubly sure by wearing boiled rubber gloves.

For the exsection I prefer using a tourniquet, and I make my incision three inches long, about three quarters of an inch to the inner side of the patella. When the synovial membrane is opened the cartilage may be inspected, and if the straight incision does not give a sufficiently comprehensive view it may be curved slightly backwards. If possible, however, it is best to be content with the longitudinal incision. The cartilage can be removed either wholly or in part, but should never be stitched. Stitching the cartilage is, or should be, an obsolete operation. If the cartilage seems to be normal and the history characteristic, and neither fringe nor loose body appear, its removal should be proceeded with. It is difficult to decide through any opening into the joint whether a cartilage is or is not loosely bound by its moorings. Removal of the cartilage in such a doubtful case is nearly always followed by a cure. Neither the surgeon nor assistant should touch the wound with anything but forceps. The sutures should be handed on forceps. The synovial membrane, capsule, and skin should be separately sutured, no blood-vessel tied, and the sterile dressings tightly applied before the tourniquet is removed. If these precautions be taken we shall probably have no anxious moments. The operation for loose bodies is not differently conducted, but great care should be taken to ascertain whether one or many bodies are present. I have on three occasions had to open up a joint a second time to remove bodies evidently present from the first.

After operation the stitches should remain in position for about ten days. The splint should then be removed and the knee massaged but not flexed. When the first fortnight has passed slight passive motions may be commenced, and in three or four weeks walking and general exercise taken. Massage of the quadriceps is a valuable aid to sound recovery.

I have dealt, gentlemen, hurriedly, and I fear most inadequately, with this important subject of derangement of the knee. May I hope that I have not altogether failed in throwing some light upon the etiology and treatment of a very troublesome ailment.

May 7th, 1906.

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SERUM THERAPY.

A Clinical Debate held under the auspices of the Chelsea Clinical Society.

DR. BALL (President) in the Chair.

Professor R. T. HEWLETT. Dr. FOORD CAIGER.
Dr. A. E. WRIGHT. Dr. BERTRAM ABRAHAMS
Dr. ALLAN MACFADYEN.

THE Chairman, in the course of a few introductory remarks, said the Society had always endeavoured to secure the co-operation of those, whether members of the Society or not, who had devoted themselves specially to the subject in question. The Society had been fortunate in securing the services of various experts in the different fields who had given the latest and most authentic views on the subject. In 1904 a discussion was held which was really on the subject of immunity, and on the present occasion it was on the allied subject of serum therapy. The Society was much indebted to those who had come to join in the debate, and he much regretted to say that Professor Sims Woodhead had wired that he was confined to the house by influenza.

Prof. HEWLETT: Mr. President and Gentlemen,—In the first place I have to thank you for the invitation you have extended to me to open this your annual debate on serum-therapy. When I see the eminent names which are to follow me I could have wished that you had chosen one of them rather than me for this task. The only consolation which I find in the course you have prescribed is a purely selfish one, for as opener I have the whole field of serum-therapy before me to wander over and to select from, unhampered and unconfined, whereas a later speaker in a debate often finds that the very points he had hoped to make most of are anticipated and fully dealt with by a previous speaker.

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I had some difficulty in deciding in what directions to choose the matter for my paper, but since this Society is eminently a *clinical* one I have thought it best to confine my remarks to data and details bearing on the practical applications of serum-therapy to treatment now and in the future, and to avoid theoretical considerations except in so far as these may be relevant to practical issues. I shall endeavour to point out some of what I believe to be the limitations of the treatment and certain directions in which we may look for advances in the treatment.

In the first place I would remind you that serum treatment has been applied in various directions:

(1) In microbic diseases, both in intoxications, in which extra-cellular toxins are freely formed and are the principal factors that required to be neutralised (the anti-toxic sera), and in infections, in which the toxins are mainly intra-cellular and the microbes themselves principally have to be attacked (the anti-microbic sera). In this connection it is to be noted that so far diseases due to bacteria have proved to be more amenable to serum-therapy than diseases due to protozoa, in which up to now little progress can be chronicled in the direction of serum treatment.

(2) In conditions in which lifeless toxic agents are derived from without, notably in snake-bites and hay fever.

(3) In conditions in which disordered activity or secretion of the tissues or glands of the body is the essential cause of the pathological condition, notably in the case of disorders of the thyroid gland.

(4) In conditions in which abnormal proliferation of cells is the essential feature, as in malignant disease generally.

It may at once be said that no other serum has anything like the therapeutic value that diphtheria serum has. The value of diphtheria antitoxin is admitted by all; perhaps the most striking testimony to it is the fall in the case mortality of the cases treated in the Metropolitan Asylums Board's hospitals from about 29·25 per cent. in pre-antitoxin days to 10·9 per cent. at the present time.

This therapeutic value of diphtheria antitoxin must be ascribed to a combination of factors which are not present in any disease other than diphtheria. Thus, in the majority of cases, and in the earlier stages at least, the disease is localised to an external, or practically external, limited site, where the toxins which cause the disease symptoms are manufactured and from which they are absorbed. In the majority of cases this local site exhibits inflammation and exudation, and the presence of membrane, whereby the disease may be diagnosed, or at least be suspected, at an early date.

It must be clearly recognised that in most, if not in all, forms of serum-therapy early treatment is of paramount importance if the disease be at all acute. No serum can repair actual tissue damage; if this exceed a certain amount before treatment is applied, no amount of the most potent serum can avert a fatal issue. The statistics of the antitoxin treatment of diphtheria illustrate this well; when applied on the first day of the disease the case mortality is practically *nil*, on the second day it is 4–5 per cent., on the third day 10–17 per cent., and on the fourth day 13–22 per cent. The early recognition of the disease, which is usually possible in diphtheria but lacking in many other diseases, is therefore one important factor, perhaps the most important factor, conducing to its successful treatment with serum.

Secondly, diphtheria is pre-eminently an intoxication produced by an excreted toxin. Now, it is well known that antitoxic sera—sera which neutralise toxins—are more potent than anti-microbic sera—sera which are bactericidal and bacteriolytic—and it is only necessary to give the proper ratio of antitoxin to toxin in order to neutralise any quantity of toxin. Such, however, is not the case with anti-microbic sera; when the infection exceeds a certain amount no quantity of the anti-serum will suffice to neutralise.

With the exception of diphtheria and tetanus, practically all other infective diseases are due to microbes which do not excrete any appreciable amount of extra-cellular toxin; the toxin is for the most part intimately associated with the cells of the infecting microbe, *e.g.* staphylococcic, streptococcic and *B. coli* infections, typhoid, plague, tubercle, etc. In the case of protozoon infections we have little evidence of the existence of any toxins, extra- or intra-cellular, and the pathogenic effects may be due partly to mechanical effects, to the results of tissue disintegration, etc., though someone, whose name I forget, has recently claimed to have obtained a toxin in malaria. This fact is unfortunate; were the majority of diseases intoxications

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from excreted or extra-cellular toxins there would probably be a somewhat better chance of obtaining efficient anti-sera, though not necessarily so. Thus the serum or antitoxic treatment of tetanus is disappointing, although tetanus anti-toxin is the most potent anti-serum that has yet been prepared. Three factors sufficiently explain this anomaly: first, the onset of tetanus is recognised only by the sequels of tissue damage—there is nothing to warn that it is about to ensue, as in the case of diphtheria, before perhaps irreparable tissue damage has been done; secondly, tetanus toxin is intensely virulent—it is the most toxic substance with which we are acquainted, and minute traces are sufficient to cause a fatal intoxication; thirdly, the tetanus toxin attacks tissues, the central nervous system, to which all other tissues are subordinated. As regards even the antitoxic sera, the most successful of the therapeutic sera, the toxicity of the toxin, its rate of absorption, the rapidity of its union with the cells and tissues, and the particular tissues attacked are all factors which must profoundly modify successful treatment with them.

When we turn to consider the anti-microbic sera, of which the anti-streptococcic, anti-plague, and anti-pneumococcic sera are types, we must confess that the results obtained with them are disappointing in the extreme. Occasionally anti-streptococcic serum acts like a charm in a streptococcic infection, but, unfortunately, this is the exception rather than the rule. As regards anti-plague serum, although favourable results have been reported by some observers, on the whole it seems to be of little or no value,* and the same may be said of anti-pneumococcic, staphylococcic, typhoid, tubercle, cholera, and other similar sera.

These anti-microbic sera, though to a small extent anti-toxic, are mainly bactericidal and bacteriolytic, acting upon the infecting and invading bacteria and getting rid of these partly by direct solution, partly by rendering them phagocytosable, and possibly also by acting as leucocytic stimulants enhancing phagocytosis. It has been shown by a series of researches† that two substances are concerned in the bacteriolytic action of an anti-microbic serum, one the complement, which is really the active agent and is a natural constituent of the body, the other the immune body, a constituent of the anti-microbic serum, and through which the complement is able to act on the bacteria, though how it does this, whether it mordaunts, prepares, or sensitises the bacteria for lysis by the complement, or acts as an actual link between bacterium and complement, is disputed.

Now, one factor limiting very materially the potency of an anti-microbic serum is not any fault of its own, but is believed to be a deficiency of the active agent, the complement, in the patient's body. The amount of complement is assumed to be limited, and part of what there is is very likely used up in the early stage of infection. Hence, although there may be plenty of immune body in the anti-microbic serum, the bulk of it is inoperative, as it has no complement to assist, complement being a very unstable body, and disappearing from the immune serum very soon after preparation. Unless, therefore, the natural complemental substances in the patient's body can be increased, anti-microbic sera must be expected to fail in anything but comparatively mild infections. A second factor of importance in the action of anti-microbic sera is this: An active anti-microbic serum may destroy all the bacteria causing the infection, but in so doing it may liberate such an amount of intra-cellular toxin that the animal, though cured of its infection, dies of an intoxication, as has been shown by Pfeiffer and Kolle for cholera and plague respectively.

There is also a third factor limiting the value of anti-microbic sera, which is this: Many, perhaps all, species of microbes vary among themselves in their attributes. That is to say, what we regard as a species, *e.g.* a streptococcus, a typhoid bacillus, etc., is really a group of closely allied varieties, all of which resemble one another in their main characters, but which differ in minuter, less apparent, but none the less highly important, ones. In fact, in all probability what we regard as the typhoid bacillus is really not a species, but is comparable to a genus among higher living forms, containing within its limits a number of species, sub-species, varieties, etc. Now, this is not obvious as regards the typhoid bacillus—it may be true only in a minor degree for this organism—but in the cases of the streptococcus, the dysentery bacillus, the *B. coli* and others, very real and apparent differences may be made out by critical

* *Vide* Bannerman's Report, 'Sc. Mem. of the Gov. of India,' No. 20.

† *See* Hewlett's 'Serum Therapy.'

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examination of different strains. Thus Gordon * has shown that streptococci apparently identical to our ordinary tests have very different chemical or fermentive activities on various carbohydrates, glucosides, and alcohols, and several races of dysentery bacilli and *B. coli* may similarly be recognised. Moreover, just as different samples of opium, cinchona bark, and strychnine beans differ both as to the kinds and as to the amounts of the alkaloids they contain, so these different varieties of what we term the same species of microbe, *e.g.* a streptococcus, differ as to the nature of their pathogenic action and of their toxins. Now, each toxin, on injection into the animal which furnishes the anti-serum, produces only its own anti-body, and therefore the anti-bodies developed by the injection, say, of one race of streptococcus may have little or no neutralising value towards another streptococcus. This fact is most unfortunate, and one that must seriously limit the possibility of manufacturing anti-microbic sera which shall be of universal value in all cases of the same kind of infection. It is now the practice to prepare anti-microbic sera with many stains of an organism; such a serum is termed "polyvalent."

I may here quote Ainley Walker's apt simile:

"If you desire to put a muzzle on your dog, you obviously choose a muzzle that will fit—one that is 'special' for your particular dog. That muzzle will probably fit with fair success a large proportion of other dogs of the same breed, and may be applicable more or less to a considerable number of dogs of other breeds in which the head is somewhat similar in size and conformation. But your sheep-dog's muzzle would not be very serviceable for a terrier, and would be quite inapplicable to a mastiff or Newfoundland. And hence, if required to muzzle all varieties of dogs, you would provide yourselves with all the varieties of muzzles necessary." †

The case of cholera comes under a category rather different from streptococcic and other infections, for the cholera vibrio in cholera is practically extra-corporeal, being confined to the intestinal tract and surface of the intestinal mucous membrane, and not making its way into the blood, tissues, or organs. The pathogenic action is therefore an intoxication, though of a kind very different from that of diphtheria or of tetanus. It is true that various investigators, notably Behring and Ransom, and Metchnikoff, Roux, and Salimbeni, have obtained a feeble toxin from cholera cultures, but the toxin inducing the choleraic intoxication is probably more correctly to be regarded, not as an excretory toxin, but as a cellular toxin derived from the breaking up or autolysis of numbers of vibrios in the intestine and absorbed by the intestinal mucous membrane. Any efficient anti-serum for cholera would therefore have to be, not simply anti-microbic, but also antitoxic, as regards this cellular toxin. This may possibly be the case, or partly so, in other conditions, *e.g.* in certain localised septicæmic processes, if such a term be admissible, possibly in localised tuberculosis, perhaps in typhoid, and in plague when the infecting bacteria are numerous.

As regards protozoa, most work has been done with the trypanosomes.

In 1902 Laveran and Mesnil * found that normal human serum caused at least a temporary disappearance of the *Trypanosoma Brucei*, the causative organism of nagana or tsetse-fly disease of horses, in inoculated mice and rats. No other normal serum possessed this power.

Laveran and Mesnil also found that the serum of sheep and bullocks which had been infected with this trypanosome, but had recovered, had curative properties for at least the goat and the sheep when infected with this species of trypanosome.

Recently Kleine and Möllers † report that by injecing an ass with the *T. Brucei* from rat infections the ass's serum acquires a distinct preventive action against infection both with the *T. Brucei* and with the *T. Gambiense* of human trypanosomiasis as tested on mice. These experimental observations, however, are very far from providing a curative serum for these diseases, and I personally do not think the outlook is very hopeful. Moreover, as many of these protozoan infections are chronic, it is quite possible that the methods of Wright may be applicable to them.

With regard to sera which have been employed

* 'Rep. Med. Officer of the Local Gov. Board for 1903-4.'

† 'The General Pathology of Inflammation, Infection, and Fever,' p. 194.

* 'Ann. de l'Inst. Pasteur,' xvi, 1902, p. 785.

† 'Zeitschr. f. Hygiene,' xxv, Pt. II, 1906, p. 229.

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to neutralise toxic agents derived from without, the chief of these are for snake-venom, for certain toxic plant proteids, and for hay fever, of which the first is of the greatest importance. The anti-sera for these poisons are of the antitoxic type, and they are just as specific in their action as other anti-sera. It is true that an anti-vernine for one particular snake-venom may have some neutralising power for a venom of a closely allied species, but if the species are unrelated there is then practically no neutralising power. Therefore a cobra-venom anti-serum is valueless against the venom of the Australian tiger-snake or of the American rattle-snake. Again, with moderate doses of a toxin, such as diphtheria or tetanus, there is always an incubation period prior to symptoms of, it may be, twenty-four hours. Snake-venom, however, acts very rapidly, within half an hour. These facts must mitigate much against any wide use of anti-venomous sera. Moreover, in order to obtain potent anti-sera, large quantities of the particular venoms are required, another practical difficulty in the way of preparing these sera.

With regard to the third group of sera I have named, those directed against morbid activity of tissues or organs, a thyroid serum for the treatment of exophthalmic goitre is the only one I need refer to. It is based on the fact that the administration of thyroid extract often aggravates the condition, the assumption being that this disease is an intoxication with the products of excessive thyroid activity. Two classes of thyroid sera have been devised; one is merely the serum of thyroidectomised animals, the *rationale* of which is based on the assumption that in health the thyroid secretion combines with something (call it an anti-body, if you like) in the body, and becomes neutralised and rendered inert, that in the thyroidectomised animal this something, this anti-body, accumulates, and on injection into the patient combines with his excess of thyroid secretion and neutralises it. A second method, recently the subject of a research by Beebe,* of New York, attempts to combat the morbid activity of the thyroid by introducing a cytolytic serum, a serum which shall have a toxic action on the thyroid cells and stop their activity. According to Beebe and Rogers, of various sera tried, one prepared by injections of thyroids removed from exophthalmic goitrous patients alone proved efficient—Rogers, however, claiming to have cured several patients with it. But if the abnormal thyroid of the disease be required to prepare the appropriate anti-serum, obviously the opportunity for the preparation of such a therapeutic serum must be very limited.

The use of cytolytic sera—sera having a destructive action on cells, might be supposed to have possibilities in the treatment of malignant disease. The injection of cells into an animal is followed by the development of anti-bodies having a destructive action on the cells used for the injection, and in this way spermo-toxins, epithelio-toxins, hepato-toxins, neuro-toxins, nephro-toxins, etc., have been prepared. In all cases, however, the sera are very specialised, if not absolutely specific, and therefore act only on the cells with which the serum has been prepared. Therefore, for malignant disease, malignant tumours would have to be the source of the curative serum.

Having now indicated some of the directions in which serum therapeutics have been, or might be, applied, I wish to make a few remarks on the practical application of serum-therapy and the preparation of sera.

As regards administration, almost everyone is agreed that the anti-sera must be injected into the body, subcutaneously, intravenously, or directly into the site where the toxins become attached, as into the central nervous system in cases of tetanus. But reports from time to time appear of the successful administration of sera by the mouth or rectum. Thus Pilcher, in a recent number of the 'British Medical Journal'* records a number of cases in which he considered oral administration to have been effective. An examination of the table he gives is, however, most unconvincing. Some four or five years ago I showed that in rabbits and guinea-pigs no trace of diphtheria or tetanus antitoxin is absorbed by the mouth or by the rectum, and it seems to me a very dangerous thing that these reports should be allowed to go unchallenged, and it is to be hoped that no one will be misled by them.

The site of the introduction of the serum is of importance. If a rapid effect is wanted and the serum is desired to bathe the whole of the tissues, undoubtedly intravenous inoculation is the proper method. In other cases the serum may be intro-

* 'Journ. Amer. Med. Assoc.,' February 16th, 1906, p. 484.

* February 17th, 1906, p. 379.

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duced at the site of infection or damage. In malignant pustule, for example, the anthrax serum should be inoculated locally; in tetanus it should be introduced into the central nervous system; if there be any particular path of absorption or infection, this may be blocked by the local introduction of the serum. Thus, in traumatic tetanus, Meyer and Ransom have shown that the toxin is absorbed along the great nerves, by the motor neurones that is, and not by the blood- or lymph-channels, and the introduction of the tetanus antitoxin in the course of the nerves is therefore indicated. In diphtheria infection an anti-microbic serum has been used locally, but principally in those troublesome cases where the bacilli do not disappear on convalescence. Tetanus anti-toxin has been used for dressing wounds, and possibly this form of application might be extended. The question of dosage is an important one. Everyone is probably aware that sufficient anti-serum must be given, but it is not so obvious that an excess must be avoided. Thus Neisser and Wechsberg found that infected animals died just as certainly if they had too much anti-microbic serum injected as if they had too little. In all probability in this instance the free immune body and complement unite, and the immune body attached to bacteria can therefore secure little or no complement, and the bacteria are not destroyed. The same holds good for anti-toxins; excess of anti-toxin tends to the production of anti-antitoxin, though in this case the objection is perhaps more theoretical than practical; still, it is a point to be borne in mind.

The question of the varying attributes of the same species of micro-organism is an important point in the preparation of sera, especially of the anti-microbic ones. Sera of this kind are now being made by injecting a number of different strains of the micro-organism, as in the case of anti-streptococcic serum, such a serum being termed a polyvalent one. It seems to me that the same principle should also be applied in the preparation of anti-toxic sera. Undoubtedly different diphtheria bacilli are found to produce varying effects on experimental inoculation; for example, one bacillus will produce more local necrosis than another. A diphtheria serum should therefore, I consider, be prepared with several strains of diphtheria bacilli and not with one only.

Again, in bad cases of diphtheria, and in the later stages, the diphtheria bacillus does not remain localised but gains access to the blood and tissues: the condition is an infection as well as an intoxication. In bad cases of diphtheria and in those coming under treatment at a late stage, I cannot help thinking, therefore, that an anti-microbic diphtheria serum, in addition to an antitoxic one, would prove of benefit and might be used with advantage. With regard to the anti-microbic sera, the lack of complement is evidently a bar to their successful use, and so far it has not been found practicable to introduce more complement. If complement be a product of the leucocytes, as seems probable, attempts may be made to increase the number of leucocytes, but again we are much in the dark respecting the utility of this. Moreover, in extensive infections we have to deal, not only with the microbes, but also with their intracellular toxins, and in such cases a serum needs to be not only anti-microbic, that is bactericidal and bacteriolytic, but also antitoxic as regards the intracellular toxins. It seems to me that it is this fact which has proved an obstacle in the preparation of efficient anti-typhoid and anti-plague sera, and it is in this direction that Dr. Macfadyen's work on intra-cellular toxins and their application in immunisation and in the preparation of anti-sera are of so much importance. In the case of tubercle and other infective granulomata, besides the microbe and its extra- and intra-cellular toxins, other factors come in. The granuloma invades and destroys the tissues and interferes with function, and later on is prone to degenerate, to break down, and to become infected with other microbes. Should an efficient serum be prepared against the microbe it could be active only in the early stages of infection; it could not replace tissue destroyed, and secondary infection would render the condition hopeless.

Again, anti-sera are only formed when the cells of the inoculated animal possess appropriate receptor groups, and it may be that animals other than the horse will be found more efficient for the production of certain anti-sera. This has been proved to be the case experimentally in several instances, and Ehrlich has suggested that animals more nearly related to man than the horse might be used to furnish the anti-serum. This principle might well be applicable in the case of diseases of the lower animals, but could only be of limited application in the case of man.

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In the case of chronic or local infections the splendid work of Wright and his co-workers has proved that we have methods of attacking the disease other than by sera. Whether the same methods will ever be likely to be efficient in acute general infections is a point which perhaps Dr. Wright might take up in the course of his remarks. As regards malignant disease, serum-therapy does not seem to hold out any great hopes. Clowes* has found that certain mice, the subjects of experimental cancer, recovered spontaneously, and that their serum actually cured other cancerous mice. But cancer is extremely specific: it can be inoculated only on animals of the same species, and, as Welch† has remarked, it is extremely improbable that the serum of any animal would have the slightest effect on a human being; a cured human being would probably have to supply the serum for human treatment.

There is one other possibility in the way of serum treatment. I have already stated that Laveran and Mesnil found normal human serum to some extent effective against the *Trypanosoma Brucei*, and cray-fish blood is effective in the mouse against scorpion venom. It may be found that certain normal sera, *i. e.* sera of untreated animals, will be effective means of treatment against certain toxins and infections, but it must be said that the prospects are not very encouraging; still, it is a line of inquiry which ought to be pursued.

In conclusion, the conviction is forced on one that serum treatment, in spite of the vast amount of research bestowed upon it during the last ten years, has not advanced much, and, unless immunisation by means of intra-cellular toxins on the lines of Dr. Macfadyen's work is successful in the preparation of efficient sera, or some principles very different from the present ones be elaborated, we cannot hope for much further advance in serum therapy.

Dr. WRIGHT.—We are met this evening to discuss together the interesting question of serum therapeutics. Professor Hewlett has already in his opening address taken you over the ground, and you will have noted that he has said little in praise of the serum therapeutics as a general principle of treatment. Not only had he little to say in the way of commendation for serum therapy as a method of combating bacterial disease other than diphtheria, but you will have noticed that much that he has said has been of the nature of extenuation and explanation of failure, coupled with suggestions for the modification of the various sera with a view to rendering them efficacious. I have felt myself as I listened in general agreement with Prof. Hewlett on the question of what has been actually achieved by serum therapeutics. On the other hand, I cannot feel that Prof. Hewlett's explanations of the failure of serum therapeutics in connection with the treatment of bacterial invasion go to the root of the matter. I have, for instance, no confidence whatever either in the practicability or in the utility of the suggestion that anthropoid apes, and generally speaking animals other than those now used, should be employed to furnish the therapeutic sera, and I am not satisfied that there is a basis of experimental fact for the theory which assumes that sera which are at present impotent would be rendered potent for good if they were exhibited in combination with them—that "complement" which is so often on the lips of bacteriologists.

I do not believe that serum therapeutics as applied to the destruction of bacteria in the body can be bolstered up by any of the devices that have been referred to. The whole system of serum therapy, except where it is a question of the neutralisation of a poison like diphtheria toxin by the aid of an antitoxic serum, appears to me to rest upon very insecure foundations.

Before attempting to make good this proposition I would like to take the opportunity of saying a word on the question of certain altogether incidental disadvantages which attach to serum therapy in all its forms, even where, as in the case of the antitoxic serum which is employed in the treatment of diphtheria, the serum accomplishes all that it is designed to accomplish.

You will immediately appreciate that I have in view those pathological phenomena, such as urticaria and articular pains, which, since they may occur after the incorporation of any foreign serum without distinction, are by the Germans very conveniently grouped together under the name of the *serum disease*.

The pathology of the "serum disease" is full of interest both from the theoretical and the practical

* 'Bull. Johns Hopkins Hosp.,' No. 169, 1905, p. 130.
† *Ibid.*, p. 147.

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standpoint. Viewed from the theoretical standpoint the disease is interesting by reason of the fact that the phenomena of the disease are, as is brought out clearly in the recent work of Pirquet and Schick,* to be interpreted as events in a process of immunisation by which the organism purges itself from the foreign serum. The serum disease is interesting also from the practical standpoint, first because there is some reason to think that the process of purgation just referred to may be associated with fundamental alterations in the anti-bacterial power of the blood; and, secondly, because the urticaria and articular pains which are to the patient sources of the most serious discomfort are largely avoidable incidents. In connection with this I showed some ten years ago † that these incidents are associated with a diminution in the coagulability of the blood, pointing out as I did so that this condition of diminished coagulability and viscidity stands in causal relation to "serous hæmorrhages" generally, and drawing attention to the fact that it is possible to restrain such serous hæmorrhages by the administration of calcium salts.

My suggestion that it would be possible by the exhibition of calcium salts to forestall the urticaria and articular troubles of the serum disease, and to cure these rapidly when they have supervened, has recently been completely justified by the work of Netter. Netter has shown in a very conclusive manner, by systematic observations conducted with controls on some hundreds of cases, that the percentage of urticarias in patients treated with diphtheria antitoxin can be very strikingly reduced—it was reduced, I think, in Dr. Netter's series of cases by four fifths—by the exhibition of calcium chloride. I would, therefore, again commend this procedure as a routine measure after serum injections, in particular in connection with prophylactic inoculations of diphtheria antitoxin. It would probably suffice in the case of an adult to administer 30 grains of calcium chloride or lactate daily from the sixth to the tenth day after the injection of serum.

Having disposed of this incidental and relatively speaking unimportant issue in connection with serum therapeutics, let me now turn to the main theme of my discourse. In serum therapy the physician proposes to adminster to the patient protective substances elaborated in the organism of an animal which has been vicariously inoculated with the appropriate bacterial vaccine. The method is commended to the physician among others by the following considerations. He believes that incidental troubles such as urticaria apart, he cannot by serum therapy do any harm even if he fails to do any good. He believes that every serum which bears the label of a reputable laboratory, stating that it is an "anti-serum" for a particular microbic infection is, as it professes to be, a serum which contains protective substances and a serum which contains these in sufficient quantity to reinforce to a sensible extent the anti-bacterial substances in his patient's blood. Further, he believes that in choosing his dose he may guide himself entirely by the clinical condition of his patient, employing more of the serum when the patient's condition is very serious, less when his symptoms are less urgent. And above all, the physician believes that he has behind him in the capacity of a scientific guardian a bacteriologist who, as a preliminary to issuing the serum, has arrived at an adequate knowledge of its composition.

The whole of this body of beliefs rests, I am convinced, upon a foundation of sand. Let me for the moment deal only with the last and most important belief, with the belief of the practitioner, that the bacteriologist who furnishes the serum has arrived at a competent knowledge of its composition. The bacteriologist will emphatically repudiate any such knowledge in connection with many of the sera which he furnishes. He will no doubt in the case of his anti-diphtheria serum, and in the case of his anti-tetanus serum, tell you the antitoxic value of the serum; for he will have measured it. But he will admit to you—if you press home your inquiries—that in the case of anti-bacterial sera, where he has not at disposal any adequate methods of examination, his sera are often sent out without any searching examination. His only warranty for labelling his sera in such a case as "anti-sera" is derived from the fact that he assumes that horses may be trusted to furnish potent anti-sera when injected with progressively increasing doses of any bacterial vaccine.

It is for you to consider whether this assumption of the serum-manufacturer is an assumption

* 'Die Serumkrankheit-Franz Deuticke,' Leipzig und Wien, 1905.

† 'Lancet,' January 18th, 1896; 'Brit. Journ. of Dermatology,' vol. viii, No. 89; 'Lancet,' September 19th, 1896.

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which he is entitled to make, and an assumption upon which you are justified in reposing confidence.

Let us try to make clear to ourselves the working theory of the bacteriologist who manufactures sera. He would probably formulate his working theory in some such form of words as the following: "If I, in a diagrammatic manner, represent the amount of protective substances in the blood of a horse anterior to inoculation by a series of dots positioned as at *a*, *a*, *a* (Fig. 1), the amount of protective substances in that horse's blood after recovery from a first inoculation of any bacterial vaccine may be represented by a dot positioned as at *b*. After second and third inoculations, conducted in each case with doses increasing by geometrical progression, the amount of anti-bacterial substances may be represented by dots positioned at *c* and *d*. Progressing in this manner, I shall arrive at a condition when there will be contained in the horse's blood very large amounts of protective substances."

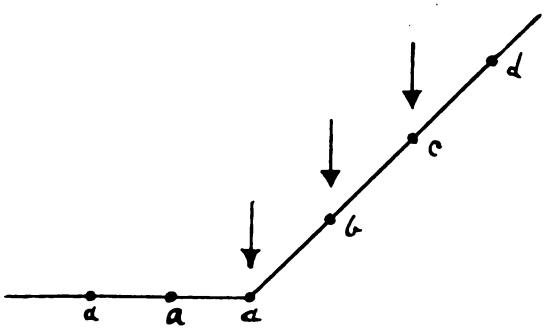

FIG. 1.

When we come to inquire into the basis upon which this comfortable theory is built up we find that results such as those just postulated are obtained in connection with the inoculation of diphtheria and tetanus toxins into horses. But let it be noted that results such as these are, even in connection with the inoculation of diphtheria and tetanus toxins into horses, not always obtained. In a certain percentage of cases no satisfactory amount of anti-toxin is ever achieved. Again, a certain number of horses succumb in the course of the treatment. Lastly, a condition may be arrived at in which injections of toxin are no longer responded to by an elaboration of anti-toxin.

It will be recognised, in view of the above, that the generalisation upon which the serum manufacturer relies is not a true generalisation from the whole of his experience in connection with diphtheria and tetanus antitoxins. It is a generalisation from his experience in connection with his successful cases. The serum-manufacturer has thus not even the justification of an argument from analogy for assuming, as he does, that the serum which he obtains by the inoculation of bacterial cultures into horses must contain protective substances. And he has even less justification in assuming, apart from actual verification, that his sera contain protective substances in sufficient concentration to be therapeutically useful. In so fundamental a matter as this the proper course for the bacteriologist is, as you will recognise, to verify everything, to measure everything, and to declare in each case the results of his measurements. In particular ought the alterations in the anti-bacterial powers of the blood which supervene upon the incorporation of bacterial vaccines into the horses which serve for the production of his sera in each case to be investigated. I take it as assured that those alterations would correspond in all respects to those which I found to occur in man in connection with anti-typhoid inoculation, and afterwards in connection with the inoculation of very many different varieties of bacterial vaccines. I may perhaps recall to your minds the sequence of events which occurs in each case, and which I have spoken of as "the law of the negative and positive phase," or as "the law of the ebb and flow and re-flow of the wave of immunity." Let me trace for you here a typical curve representing the changes in the anti-bacterial power of the blood which occur after an ordinary inoculation (Fig. 2 A).

This form of the curve may be modified. Here, for instance (Fig. 2 B), where only a small dose of vaccine is administered, the negative phase is elided, and duration of the positive phase is shortened. Again, where a large dose is inoculated the negative phase is accentuated, until finally the positive phase is abolished, the blood remaining for a considerable period below the level at which it originally stood (Fig. 2 C).

The significance of these curves in connection with serum therapeutics will immediately come home to you. It will be appreciated that according as a larger or smaller dose of bacterial vaccine has been incorporated into the particular horse, and according as an earlier or later date may have been chosen for the bleeding of that horse, his blood will contain, in the one case, unneutralised

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bacterial poisons and less protective substances than the normal blood, and in the other case protective substances in excess of those found in the normal blood.

In the curves I have drawn (Fig. 2) I have set forth, as you will have observed, only the events which supervene upon a single inoculation. Let me now, seeing that we have in serum therapy to deal with sera which are obtained after a whole series of inoculations, pass on to consider with you the results which may be achieved by a series of inoculations.

Here one of three things may happen. (1) Each inoculation may operate as an independent event,

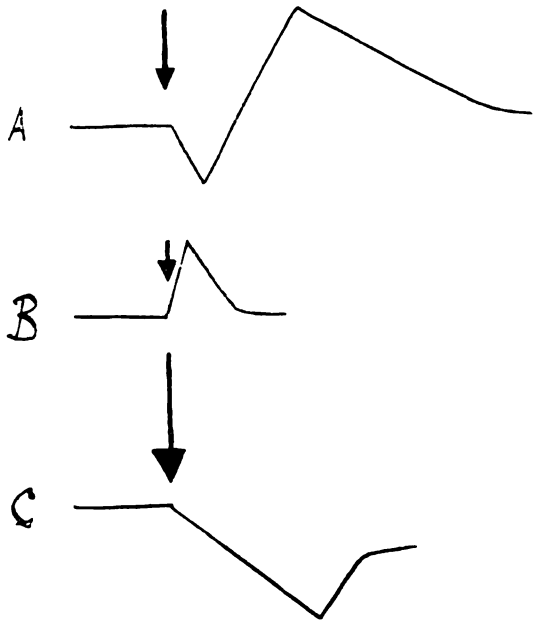

Fig. 2.

as indicated in Fig. 3 A; (2) the inoculations may produce a cumulative effect in the direction of the negative phase, as indicated in Fig. 3 B; (3) the inoculations may, as in Fig. 3 C, produce a cumulative effect in the direction of the positive phase.

The first result is, I believe, the ordinary result of inoculation of a bacterial vaccine when the animals are allowed to recover completely between the successive inoculations.

The second result is, I suspect, very frequently achieved when the inoculations are pushed.

The last effect would, if I may argue from a very large experience of the effects of inoculation on man, appear to be only exceptionally realised. Moreover, even where the very best results are achieved in connection with the elaboration of anti-bacterial substances in the organism, that miracle of successful immunisation which accomplishes itself in connection with the inoculation of diphtheria toxin and tetanus toxin into horses—a miracle which gives us, often within the compass of a very few cubic centimetres of serum, a quantum of anti-toxin which suffices to alter the whole course of the patient's disease, is, so far as we know elsewhere, never even distantly approached.

You will recognise how different all this is from the dream which you share with the serum-manufacturer. The serum-manufacturer makes his

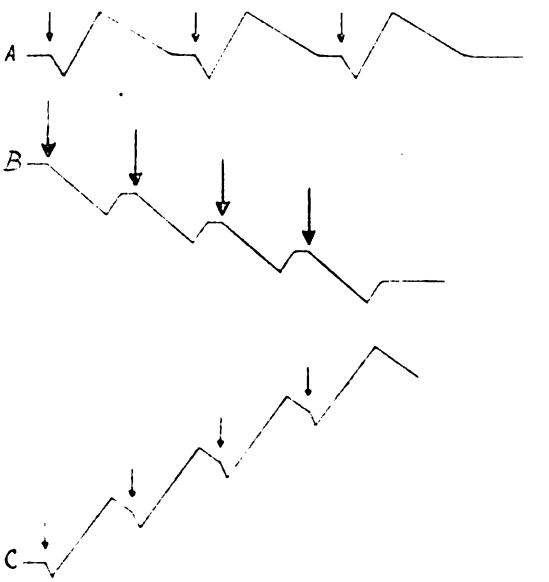

Fig. 3.

serum, and you administer it thinking that beyond doubt you are administering a quantum of anti-bacterial substances which will count in your patient's conflict with his bacterial invasion. And all the while the product which the serum-manufacturer is sending out, and which you are administering, may not contain any appreciable quantity of protective substances.

That serum may, on the contrary, contain, practically unaltered, the identical bacterial poisons which were originally incorporated into the horse. And by the agency of these it may so happen that you may, as by the incorporation of a dose—here an unmeasured dose—of a bacterial vaccine, be

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lowering, temporarily at least, your patient's powers of resistance with respect to the micro-organisms with which he has to deal.

In connection with this last possibility, two questions immediately press for solution. The first is the question as to where the responsibility rests if a serum which contains a toxic bacterial element is exhibited as a serum which contains protective substances. The second is the question as to whether there are any stigmata by which the bacteriologist, or failing him, the clinician, would be capable of discriminating between a serum which contains protective substances and a serum which contains toxic bacterial elements.

In particular, the issue as to where the responsibility rests in the case of the administration of a serum which is something quite other from what it is assumed to be is an issue which must come home to us all. We may take it that the physician will contend that the responsibility lies on the shoulders of the bacteriologist, and will point to the fact that the serum is labelled as an "anti-serum" as a fact which places the bacteriologist clearly in the wrong if the serum does not contain protective substances in appreciable quantity, and above all, if it contains toxic bacterial elements.

The bacteriologist, for his part, will, no doubt, contend that when he has incorporated into the horse his bacterial vaccines, and when he has waited after the last inoculation until the horse is "restored to health" he has fully discharged his responsibility, and that the clinician must be answerable for any results which may follow from the exhibition of the serum.

It may be debatable as between physician and the bacteriologist as to who is to take the blame, but it is for us to look at the matter from the patient's point of view. From the point of view of the patient someone is assuredly to blame if a serum is administered which is quite other from what it is intended to be, and assuredly, also, we must hold the practitioner who administers the serum as responsible to the patient.

If I am correct in this, the physician should abstain from assuming with regard to his patient responsibility for any serum except where, as in the case of an anti-diphtheria serum, the bacteriologist is prepared to give him definite information with respect to the amount of protective substances in the serum. Again, all experimentation with sera of uncertain composition ought, I submit, to be undertaken exclusively by those who, understanding the possibilities of error, would proceed in these matters with a due sense of responsibility. I shall return to the subject later.

For the present, let me turn to the second of the issues spoken of above, and let me consider with you whether there is any possibility of determining in connection with a serum which purports to be an anti-bacterial serum whether it is rich in anti-bacterial elements, or whether it contains toxic elements derived from the bacterial vaccines which were inoculated in the horse.

There are in point of fact a whole series of methods which may be exploited in this connection:

(1) It may be determined upon animals whether a serum which purports to be an "anti-serum" does or does not possess toxic properties.

In this connection attention may be drawn to the fact that a particular "anti-staphylococcus serum" which was examined by Dr. Bulloch was found to be extremely toxic.

(2) It may be determined upon animals whether, failing the production of an overt toxic effect on animals, the incorporation of a serum reduces the resisting power of the animal organism to the corresponding bacterial infection.

In this connection attention may be drawn to the fact that one of the "anti-plague sera" which was furnished to the Indian Plague Commission for trial upon man, and which fell to me as a member of that Commission to test, proved to be a serum which, while it was not definitely toxic in the sense that the anti-staphylococcus serum referred to above was toxic, was none the less a serum which lowered the resisting power of guinea-pigs to plague infection, causing these animals to succumb to the test-inoculations in shorter time than the control animals—the extent to which death was hastened being in each case directly proportional to the dose administered.

(3) It may be determined whether the addition of a serum to normal human serum does or does not deprive this serum of its anti-bacterial powers. I may here instance the fact that a highly agglutinating "anti-typhoid serum" which was suggested for use upon the troops in South Africa proved to be a serum which could not be added even in small

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quantity to normal blood *in vitro* without abolishing the bactericidal power which is normally exerted upon the typhoid bacillus by human serum.

(4) Lastly, a serum which purports to be an anti-bacterial serum may be tested by the method which finds illustration in the curves below.

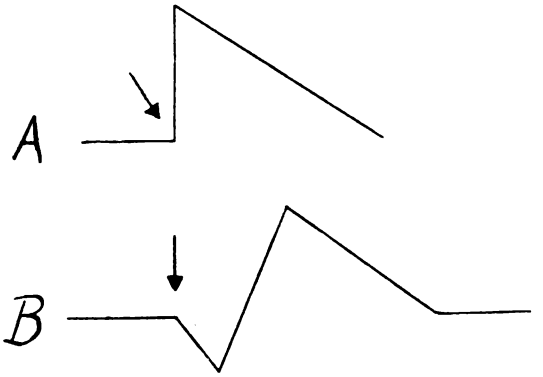

FIG. 4.

The curve which I have labelled A would, I take it, be accepted by everyone as the type of curve which we are entitled to expect when we make a series of measurements of the protective substances of an animal's blood in connection with the intravenous injection of an anti-serum.

The curve which I have labelled B would, I take it, be conceded to be the type of curve which would be obtained in connection with the intravenous inoculation of a bacterial vaccine.

The inference would appear to be justified that where the latter instead of the former type of curve is obtained in connection with the inoculation of a serum that serum must contain, not anti-bacterial substances, but bacterial elements derived from the vaccine which was employed in the "immunisation procedures."

I have applied this test in connection with a serum which was derived from a reputable laboratory, and which was labelled "anti-tubercular serum," with the result that I obtained in the case of a rabbit inoculated with this serum a curve of the type B—a curve which was practically identical with that obtained in the case of a control rabbit inoculated intravenously with tuberculin. I may point out to you that this observation falls into line with the fact that typical "tuberculin reactions" have over and over again been recorded in connection with the exhibition of "anti-tubercular sera" to human tubercular patients. You will in view of this, I think, be with me when I urge that not alone such experiments as the above on animals, but if possible also measurements of the effect exerted on the anti-bacterial power of the human patient, ought preliminary to its issue to be made in connection with every serum issued for the treatment of septicæmic disease. Where such definite measurements of the effect exerted by a serum upon the blood of patients have not been carried out, it is sometimes practicable to discern the character of the serum from clinical observation alone.

(1) The clinician would be justified in suspecting the presence of bacterial poisons in a serum in those cases where a tuberculin reaction or other similarly characteristic reaction supervened upon the exhibition of the serum.

(2) He would perhaps be justified in a similar suspicion where severe constitutional symptoms, other than those which are characteristic of the serum disease, supervene upon inoculation—in particular where severe symptoms of intoxication make their appearance with greater frequency in connection with the treatment of the graver cases than in connection with the less serious cases of the disease.

(3) Lastly, the clinician would be justified in suspecting the presence of bacterial elements in serum where there supervenes immediately upon its inoculation an exacerbation of patient's symptoms, followed after an interval by a distinct improvement.

This particular sequence of events would, I submit, find its most satisfactory explanation in the hypothesis that the serum is functioning as a vaccine.

The idea that a serum may in reality be a bacterial vaccine in disguise, and the idea that benefit may be derived from the inoculation of a bacterial vaccine in cases of septicæmia are new and as yet unventilated ideas. I am anxious to commend these ideas to your consideration. Let me begin by putting it to you once again that, in view of all that we know with respect to the difficulty of obtaining a cumulation of anti-bacterial substances in the blood, and in view of what we know with respect to the rule of thumb manner in which the immunisation of horses is at present conducted, it is quite as likely that a residuum of the bacterial vaccines originally incorporated should be contained in the serum issued as that a really useful quantum of anti-

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bacterial substances should be contained in it. If this is so a serum obtained by the immunisation of horses, while it may at one time be a vehicle for anti-bacterial substances, may at other times quite well be in effect a bacterial vaccine.

Passing to consider the possibility of bacterial vaccines rendering useful service in connection with the treatment of septicæmic infections, I have to confess that the idea that bacterial vaccines could here play a useful *rôle* was only a short time ago very uncongenial to my preconceived notions. I conceived that when bacteria found access to the blood and generalised themselves in the system the machinery for immunisation which is at the disposal of the organism was fully called into action. In accordance with this I assumed that to inoculate bacterial vaccines in such circumstances would be to add fuel to the fire without contributing anything to the elaboration of those anti-bacterial elements which serve to extinguish the conflagration.

I have now recognised that both the premises upon which I built and my inferences from those premises are assailable.

In the first place, while it is true that the machinery for immunisation with which the organism is provided is called into action in septicæmic diseases, it would seem certain that under the conditions which obtain in such diseases that machinery is often not working to its full capacity.

An unmistakable indication of this is furnished by the fact that in the exactly analogous conditions which obtain where bacterial cultures or the filtrates from these are inoculated intravenously into horses there is obtained, in some cases at least, only a very poor yield of protective substances. These observations on horses fall into line with observations recently made by Douglas and myself upon men in connection with cases of Malta fever and in connection with a case of infective endocarditis which will be further referred to.

Again, in the reasoning which I above rehearsed the possibility of a different effect being produced by bacterial elements introduced into the blood-stream and the same bacterial elements introduced directly into the tissues was overlooked. Yet consideration will show that there may quite be important differences, first in the matter of the toxic effects exerted, and secondly with respect to the immunising response elicited, by one and the same quantum of bacterial elements introduced directly into the blood-stream, or as the case may be directly into the tissues. The general intoxication effect—which above all we have to apprehend in septicæmic conditions—may be expected to be greatest where, as occurs in these infections, bacterial derivatives find direct access to the circulating blood, and least where, as would be the case in the inoculation of a vaccine, the bacterial elements are introduced into the tissues. In this latter case, as may often be seen in connection with the inoculation of small quanta of anti-typhoid bacilli when the patient keeps his bed after the inoculation, the toxic effect of the vaccine may expend itself exclusively upon the tissues at the seat of inoculation, constitutional symptoms being here practically absent.

Equally important are the differences which may manifest themselves in the matter of the immunising response according as one and the same quantum of bacterial elements is incorporated into the blood, or, as the case may be, directly into the tissues. In the case where bacteria are, as in septicæmic conditions, found in the blood-stream, or in organs standing in direct relation with this, the bacterial derivatives are of necessity diluted by the whole volume of the blood and lymph before they can come into application upon the tissues in which, we may take it, the machinery for the elaboration of protective substances is located. In conformity with this great dilution of the bacterial derivatives a comparatively speaking ineffective immunising stimulus will here be administered. In contrast with this where a bacterial vaccine is inoculated directly into the tissues the bacterial products will come into application upon these in a very concentrated form, calling forth a correspondingly larger production of protective substances.

Such larger production of protective substances is in point of fact regularly achieved in the horse in connection with the production of diphtheria antitoxin, when in lieu of intravenous inoculations, subcutaneous and intra-muscular inoculations are resorted to and, it would seem, in particular in the case where the inoculations are made with very concentrated toxins. My fellow-workers and I have also recently, in connection with the treatment of a considerable number of cases of Malta fever, and in connection with one case of infective endocarditis—the details of which will be elsewhere published—achieved by the exploitation of

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the corresponding vaccines in carefully controlled doses both an increased production of protective substances, and in each case in association with this a rapid cure.

Having now, I hope, made it clear that a serum which is administered as an "anti-serum" may in reality be a bacterial vaccine in disguise, and having further indicated to you that bacterial vaccines may be exploited with very happy results in connection with the treatment of septicæmic disease, I think that you will bear with me when I suggest to you that many of the successful results which have been achieved by serum therapy would find their simplest explanation in the assumption that the sera which were exploited operated as bacterial vaccines.

The idea that this might be so suggested itself to me first in connection with a particular sample of highly agglutinating anti-Malta fever serum which was prepared and issued by me from the Pathological Department of the Army Medical School, Netley. It was reported to me with respect to this sample of serum that it produced in the two Malta fever patients upon whom it was employed in each case, as its first result, an alarming exacerbation of the fever, and in association with this in each case very severe urticaria and œdema. After the lapse of a few days there supervened a sudden turn for the better, resulting, I understand, in each case in a rapid cure.

Looking back on these cases and calling to mind on the one hand the fact that this sample of serum was obtained only after the horse had been subjected to a long course of what was throughout in intention a process of immunisation; and on the other hand, the fact that nothing at all resembling the effects which were obtained by this sample of serum had been obtained with samples drawn off earlier in the course of the horse's "immunisation," which were tested upon Malta fever patients—among others myself, at a time when I came under that category—and correlating with these facts the fact that an exacerbation of the fever is seen also in connection with the inoculation of a Malta fever vaccine into Malta fever patients, I regard it as in the highest degree probable that the serum which caused the effects described above must have contained toxic elements derived from the Malta fever vaccines which had been incorporated into the horse.

I have seen a train of events somewhat similar to that recounted above supervene also in connection with the inoculation of antistreptococcus serum, and I suspect that some of you may also have had similar experiences, for I gather from the literature that intoxication symptoms followed by improvement in the patient's condition are from time to time met with in connection with the inoculation of many different "anti-sera."

Isolated facts cannot, however, furnish a basis broad enough for a generalisation such as that which I am seeking to commend to your acceptance. You will, I doubt not, as a precondition to embarking upon any serious consideration of the hypothesis I have suggested to you, desire to be furnished with a summary of the best results which have been obtained in connection with the serum therapy of septicæmic diseases; and you will desire to have furnished to you, in connection with that summary, data which would countenance the idea that the results obtained may properly be referred, not to protective substances, but to toxic bacterial derivatives contained in the serum employed.

It so happens that in a paper recently published by Chantemesse on the treatment of typhoid fever by an "anti-typhoid serum" I find as nearly as possible all that I require for compliance with your wishes. Let me place you in possession of the facts which are recorded in Professor Chantemesse's paper. He has during the last five years treated with his "anti-typhoid serum"—if I understand aright—all the cases, 712 in number, which have been admitted to his typhoid wards. By placing side by side with the results obtained upon these patients the results obtained without serum in the typhoid wards of all the other Paris hospitals taken together, the following comparison is obtained:

	Number of cases of typhoid fever treated.	Number of deaths which occurred among these.	Case mortality.
			Per cent.
Treated without serum . . .	3595	753	17·3*
Treated with serum . . .	712	27	3·7

* The arithmetic would here appear to be at fault.

It cannot be doubtful to anyone who considers these results, or who has had the privilege, as I have had, of visiting Prof. Chantemesse's wards

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and seeing his cases, that we have here a very brilliant and, I presume, an absolutely unique achievement in connection with the treatment of typhoid fever.

Our present concern in connection with that achievement is to consider whether the results which have been obtained are to be imputed to protective substances elaborated in the organism of the horse and transferred to the patient in the vehicle of the serum, or to a residuum of the typhoid vaccine originally inoculated into the horse and transferred to the patient in the vehicle of the serum.

This fundamentally important issue is not discussed in M. Chantemesse's paper. We are, however, supplied with certain data from which we can make our own deductions.

Let me try to marshall these data for you:

(1) We learn in M. Chantemesse's paper that two well-marked phases come under observation in connection with the inoculation of the serum. Immediately after inoculation there supervenes a "phase of reaction." This phase may last from four to five days. It is associated with an exacerbation of the patient's symptoms.

Upon this phase of reaction follows a "phase of defervescence."

There would seem to me to be little doubt that these two phases must be identified with the "negative" and "positive phases" considered earlier in this paper. In other words, it seems to me clearly indicated here that the inoculation of the serum is followed by a reduction in the anti-bacterial power of the blood—a reduction which stands in relation to the exacerbation of the fever—and that this is followed up by an increase in the anti-bacterial power of the blood, standing in relation with the defervescence which is observed.

(2) We learn that the dose of the serum which is employed is very small—"a fraction of a cubic centimetre"; further, that this dose is reduced proportionally with the severity of the symptoms and the gravity of the case. It may even, I understand, be reduced in very severe cases to "half a drop."

Again, we learn that when re-inoculation is resorted to the dose is further cut down by one half; and, lastly, we learn that where re-inoculation is undertaken it is undertaken only after the expiration of a considerable number of days—in the case of the published curves after twenty-three and sixteen days respectively.

When we come to ask ourselves whether this scheme of dosage, a scheme of dosage which has given the brilliant results above recorded, can be reconciled with the idea that we are dealing here with a true "anti-typhoid" serum, it becomes clear as noonday that the scheme of dosage employed is the exact reverse of that which would be apposite if we were dealing with a true "anti-serum" such as anti-diphtheritic serum.

When dealing with a true anti-serum one does not limit the dose in an ordinary case to "a fraction of a cubic centimetre," nor does one diminish the dose to "half a drop" in the gravest cases—those cases where the organism stands most in need of protective substances. Rather does one, where a true anti-serum is to hand, give in each case, unless where limited by financial considerations or considerations which have reference to the serum disease, large doses of serum. Again, in the more serious cases one lets all the considerations above referred to go by the board and gives larger and larger doses. And, yet again, where the result which one desires is not achieved by a single inoculation one does not allow a long interval to elapse before re-inoculation.

In contrast to all this when one is exploiting a bacterial vaccine in the treatment of a septicæmic disease one would, except for the fact that the dose can much more appropriately be regulated by the measurement of the anti-bacterial power of the patient's blood, follow a scheme of dosage which would be an exact replica of that followed by M. Chantemesse. I think I need not further labour my point.

(3) Let me turn to a further series of considerations which suggest that the anti-typhoid serum which M. Chantemesse has so brilliantly exploited is a typhoid vaccine in disguise.

It is, as all who have attempted it know, a very difficult task in the case of the horse to carry immunisation against the typhoid bacillus up to the point at which a satisfactory "anti-serum" is produced. There is thus an *à priori* improbability that M. Chantemesse has obtained in his serum a therapeutically useful quantum of protective substances. Nor does M. Chantemesse, apart from the fact that his serum is efficacious in reducing the death-rate in typhoid fever, claim very much for his serum. What he claims for it amounts to no more than this—that the serum possesses a con-

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siderable agglutinating power, and that a quantum of serum corresponding to the maximum dose which is employed therapeutically upon man—that dose, if I understand M. Chantemesse aright, never exceeds "a fraction of a cubic centimetre"—may with impunity be inoculated intra-cerebrally into rabbits and guinea-pigs.

I would ask you to note that neither the one nor the other of these findings is in any way inconsistent with the idea that the serum may owe its efficacy to the presence of typhoid vaccine.

The presence of agglutinating elements in the serum cannot, as we have seen, be accepted as evidence of the absence of bacterial derivatives. You will remember that the toxic "anti-Malta fever serum" which was discussed above was possessed of well-marked agglutinating power. And you will remember also that the "anti-typhoid serum," referred to above as a serum which completely abolished the bactericidal power which the normal blood exerts upon the typhoid bacillus, was in like manner a serum which possessed a well-marked—indeed, an altogether exceptionally well-marked—agglutinating power.

If the presence of agglutinins cannot be accepted as evidence of the freedom of a serum from toxic properties, much less can in the case of an "anti-typhoid serum" experimental results such as those obtained by M. Chantemesse, on rabbits and guinea-pigs be accepted as adequate evidence. These I regard as quite inconclusive, first, because very marked constitutional symptoms may be produced in man by doses of typhoid vaccine which are insignificantly small in comparison with those which those which are required to kill rabbits or guinea-pigs; and secondly, because, as pointed out earlier in this paper, the resistance of the organism to bacterial invasion may be seriously reduced by the exhibition of the serum, even where ostensible symptoms of poisoning are completely absent.

Scrutinising M. Chantemesse's successful results from all the different points of view which I have asked you to consider, I think there is no way of escape from the conclusion that his results—and I would urge that these are the only statistical data which are conclusive as to the utility of serum therapy in connection with septicæmic disease—are in all probability to be regarded as results obtained by the inoculation of bacterial vaccines.

Let me try now to bring out in clear relief the salient points I have endeavoured to establish in this discourse.

(1) I have tried to make good to you the proposition that the sera which are issued as "anti-sera" for the treatment of septicæmic conditions may, as the case may happen, contain a certain quantum—not necessarily a therapeutically useful quantum—of protective substances elaborated by the horse, or they may be inert, or finally they may contain bacterial elements derived from the culture originally inoculated into the horse.

Any one of these three varieties of serum may at present be sold to you under the denomination of an "anti-serum."

(2) I have drawn your attention to the fact that the scheme of dosage which would be indicated in the case of a serum which contained only protective substances would, if followed in the case of a serum such as was last in question, be dangerously erroneous. In like manner a scheme of dosage such as would be appropriate in the case where the serum is the equivalent of a bacterial vaccine would, in connection with a true anti-serum, be absolutely ineffective.

(3) Lastly, I have endeavoured to bring home to you that there is good reason to believe that the only brilliant results which have been achieved by serum therapy on a series of cases—to wit, the results of M. Chantemesse—have been achieved by the inoculation of a serum which may be presumed to have functioned as a bacterial vaccine.

If useful serum therapy in septicæmic diseases resolves itself into a question of the inoculation of bacterial derivatives, there is no need, I think, for me to point out to you the moral.

(To be concluded.)

We have received for examination and report samples of malted cocoa and milk chocolate from Messrs. J. S. Fry & Sons, Ltd. The malted cocoa can not only be accurately described as constituting in itself a perfect food, but what is very important, it can also be justly recommended for the reason that care has been taken to make it appetising as well as digestible. This malted cocoa is an excellent dietetic preparation, giving first-class results when tested by theoretical or practical standards. In milk chocolate the practitioner will find a wholesome sweatmeat that he can commend for use, especially when its value as a food is taken into consideration.

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A LECTURE

ON

FIBROIDS AND OVARIAN CYSTS COMPLICATING PREGNANCY, PARTURITION, AND THE PUERPERIUM.

By AMAND ROUTH, M.D., B.S., F.R.C.P.Lond.,

Obstetric Physician to Charing Cross Hospital, and Lecturer in Midwifery to the Medical School; Consulting Physician to the Samaritan Free Hospital for Women and Children.

GENTLEMEN,—Pregnancy and parturition are normal processes, but under various abnormal conditions, local or general, these physiological processes may bring a woman into grave danger and tax greatly her medical attendant's professional resources.

Among the local causes which may cause danger to a woman during pregnancy or labour are certain tumours, and I propose to-day to discuss two groups of tumours which are the most commonly encountered, uterine fibroids and ovarian cysts.

I, FIBROIDS.

(1) *Complicating Pregnancy.*

Diagnosis.—The diagnosis of fibroids complicating pregnancy can usually be made if the doctor's attention be drawn to the case, but unless he makes a rule of examining all women who wish him to attend them in their approaching confinement, or unless the woman's complaint of pressure symptoms or discomfort cause him to examine her specially, he may not become aware of the tumour till labour sets in.

In early pregnancy fibroids stand out from the uterine body and are easily recognised. Their presence, however, obscures the evidence of pregnancy, and oftentimes no "certain signs" are obtainable till the sixth or seventh month of gestation.

After seven months the fibroids soften and become flattened out as Cullingworth and Depaul

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have pointed out (*assouplissement*), and fœtal movements, fœtal breathing, and the fœtal heart may be thereafter detected. Indeed, in the last few weeks of pregnancy abdominal fibroids may be overlooked altogether, so great is their *assouplissement*.

(*a*) *Growth of fibroids during pregnancy.*—There is usually some growth of fibroid tumours during pregnancy, but the pressure symptoms which may be present are often due to the abnormal size of the uterus rather than to increase of the individual fibroids. Fibroids near the placental site grow more rapidly than those at a distance.

(*b*) *Pressure symptoms.*—Pressure symptoms are not uncommon if there is a pelvic fibroid. There may be only a general restlessness, with insomnia and neurosis, or there may be purely local symptoms, such as difficulty of micturition owing to pressure on the neck of the bladder or the urethra, or there may be frequency of micturition by pressure on the top of the bladder.

Difficulty in defæcation or irritability of the rectum may also be present, and often dyspepsia, flatulence, nausea, or vomiting. It is rare, however, that suitable treatment will not alleviate the pressure symptoms.

Until you have had cases of this type, where the pelvic fibroid has been replaced above the brim either spontaneously, or by your assistance, you will hardly credit the fact that the vast majority of these cases need no surgical treatment at all, from the obstetric point of view, though the fibroids may need attention afterwards.

The following case, one out of many where a palliative treatment was adopted, bears out my contention.

Case 1.—Mrs. C. L.—, seen in consultation with Dr. O'Meara, of Tottenham, in June, 1905. Æt. 33 years. Had been married six years, and had never been pregnant. Had seen nothing for three months. Complained of a growing lump in abdomen, with increasing difficulty in micturition and occasional vomiting.

On examination, the patient was found to have a large, hard, nodular fibroid reaching to the navel and another large nodular mass was filling the pelvis. The os uteri was out of reach above the pubes. The pelvic fibroid was so immovable that it seemed impossible to suppose it would rise above the brim, and so allow labour (for I suspected a three months' pregnancy to be present) to occur normally in six months. With the experience, however, of many similar cases, I advised the doctor to temporise, and we ordered the patient to remain in bed, and to take saline purgatives freely. She soon improved, the pelvic tumour gradually rose, and some weeks afterwards I found the pelvic fibroid was above the brim, and the os uteri was within reach. I was able to push up the fibroid still more, and the os uteri then assumed a normal position. The fœtal heart and fœtal movements could then be felt. Subsequently Dr. O'Meara wrote that in December, 1905, he had safely delivered his patient of a living child by forceps, the presentation being an unreduced occipito-posterior position of the vertex; there was no hæmorrhage, the fibroids did not obstruct, and the patient made a good recovery, involution being good. The fibroids, of course, are still present, though the doctor tells me that one of them is very much smaller.

Case 2.—A somewhat similar case was recently in Golding Ward; a married woman, æt. 35 years, had a fibroid on each side of the supra-vaginal cervix, bulging downwards into the pelvis, displacing the cervix upwards almost out of reach. I was able to push up one fibroid under anæsthesia, and some weeks afterwards, when she was again admitted, we found both the fibroids well above the brim, so that Cæsarean section will probably not be needed. She is now about seven months pregnant, and reports herself to me every two or three weeks. [*N.B.* She has since been spontaneously delivered at full term.]

(*c*) *Do fibroids lead to sterility or premature labour?*—Statistics and experience go to show that pregnancy occurs about as often in married women of 35 years of age who have fibroids as in those who have not, so that the almost universal idea that fibroids cause sterility cannot well be maintained.

Statistics given by Lefour, Nauss, Kelly, and Pinard show also that in 1174 pregnancies complicated by fibroids only 264 aborted, *i. e.* 21·6 per cent., which is just above the normal proportion of abortions, which may be taken as from 18 to 25 per cent. It is probably true, however, that labour comes on a week or two before full term in a considerable proportion of these cases (Herman).

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(2) *Fibroids complicating Labour.*

It is now, as I have said, well established that fibroids rarely obstruct labour, whatever their position may be in the early months of pregnancy. There are two exceptions to this, viz. where the fibroid is cervical, or where it is intra-ligamentous, burrowing in the broad ligament, and therefore in both cases unable to be elevated above the brim of the pelvis.

In most other cases the fibroids, which at three months of gestation may appear to block the pelvis completely, or which perhaps displace the os uteri, so that it cannot even be felt *per vaginam*, are gradually or suddenly raised out of the true pelvis, and come to lie altogether above the parturient canal. This displacement is the result of the combined *assouplissement* of the fibroid, traction by the growing uterus, and counter lateral pressure by the presenting part of the fœtus.

Apart from the small percentage of cases where labour is obstructed by fibroids, labour may be influenced disadvantageously in other ways. Amongst these may be mentioned fœtal mal-presentations, post-partum hæmorrhage, and sub-involution.

	Vertex.	Breech.	Transverse.	Prolapse of Limbs.	Prolapse of Cord.	Placenta prævia.
With fibroids . .	52 per cent.	25 per cent.	20 per cent.	4·7 per cent.	6·8 per cent.	3·5 per cent.
Normal percentages	93·6 ,,	3 ,,	1·16 ,,	0·9 ,,	0·44 ,,	0·01 ,,

In a large number of cases of fibroids complicating pregnancy which I have been able to collect I have calculated that mal-presentations are largely in excess.

Again, there is no doubt that post-partum hæmorrhage is more likely to occur when fibroids are present, owing to the tendency of the placenta to be implanted low down and, in some cases, to be unduly adherent owing to a co-existing endometritis. The uterus, too, does not contract and retract so efficiently, and a rather free oozing may persist from the placental site for some days after labour.

(3) *Fibroids complicating the Puerperium.*

Involution is retarded but not prevented. "After-pains" occur more frequently, and the lochia is increased in quantity and lasts longer. Intra-mural fibroids may atrophy during involution and have been known to disappear, but they usually tend to be extruded and to become more subserous. More rarely they become more submucous and may even be extruded bodily into the uterine cavity. If the uterus becomes septic, an intra-mural fibroid, and still more likely a fibroid which has become submucous, is liable to become infected, and a fatal result from peritonitis or septicæmia is not unlikely.

The following is a case in point:

Case 3.—Miss A. R—, æt. 40 years. January, 1882: Large irregular tumour to navel. Amenorrhœa four months. Diagnosis: Four months' pregnancy in a fibroid uterus. There was a pedunculated subperitoneal fibroid at fundus and three subperitoneal fibroids on the anterior uterine wall, but the pelvis was free. The patient had also well-marked secondary syphilis, dating from the time of her impregnation. The patient went to full term, and was delivered by her local doctor by means of forceps. On the third day she had a rigor and a temperature of 105° F., and when I saw her again, three days later, a putrid, stringy fibroid, the size of a large orange, was found *in utero* and easily removed, but she did not rally, and died on the twelfth day of acute septicæmia.

With our present knowledge this life might have been saved by hysterectomy if done when sepsis had first declared itself.

Occasionally a fibroid will slowly degenerate after labour, partly as a result of bruising during the labour, and partly as a result of uterine thrombosis interfering with its venous circulation. Such fibroids will cause peritonitis, and as they may also readily become secondarily infected from a septic endometritis, they may in either case lead to a fatal result.

Treatment.—There are gynæcologists in London and elsewhere who look at these cases from a purely surgical point of view, and advise and prac-

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tise hysterectomy in the early months of pregnancy for the relief of the current pressure symptoms; but our aim ought to be to save the mother from as much risk as possible whilst giving her every chance of having a living child, which, from the circumstances of the case, may be the only child she is likely to have.

I have pointed out how rarely fibroids obstruct labour at full term, and our object should be to tide the patient over the earlier months, to lessen the pressure symptoms by rest in bed, by saline purgatives, and by trying to encourage the fibroid to rise out of the pelvis.

The patient should lie with the shoulders low and the feet of the bed raised on blocks about eight inches high.

Sometimes the pelvic fibroid can be elevated, with or without anæsthesia, by digital or hydrostatic pressure in the Sims' or knee-chest position.

Induction of abortion for a fibroid likely to obstruct labour is never justifiable. This was the treatment thirty years ago, when Cæsarean section, with its 79 per cent. mortality, was the only alternative. Owing to the obstructing fibroid we cannot efficiently empty the uterus if abortion is induced, and the risk of sepsis is therefore very great. The more difficult it is to induce abortion, the more dangerous is the proceeding; the easier it is, the less necessary is it to interfere at all.

In the absence, therefore, of really urgent symptoms, adopt an expectant attitude, for two lives are here at stake.

Supposing, however, that the case is seen in the early months of pregnancy, *before fœtal viability*, and it is considered best to interfere at once, we can do one of two operations, myomectomy or hysterectomy. Myomectomy is indicated when the uterine fibroids are pedunculated, or if sessile, few in number, not deeply imbedded, and not too broadly attached to the uterus. Myomectomy leaves the child untouched and gives the patient the chance of subsequent pregnancies. Statistics show that 6 per cent. of the patients abort if the fibroid removed is pedunculated and about 40 per cent. if the tumour is sessile. The mortality of the operation is high, about 16 per cent. Hysterectomy, before fœtal viability, is required when intractable hæmorrhage or sepsis follows an incomplete abortion in cases where, owing to obstructing fibroids, the placental site cannot be reached.

It is also required if pressure symptoms are really severe, in cases where myomectomy is impracticable, and after every effort has been made to elevate the obstructing fibroid.

In all other cases hysterectomy should be postponed till the child is viable. The mortality of hysterectomy under these circumstances is small, perhaps 8 or 10 per cent.

If the patient be first seen *after fœtal viability*, and if all attempts at elevation of the obstructing fibroid have failed and obstruction is certain, Cæsarean section is necessary to save the woman and the child. As a rule in such a case it is better to follow this at once by hysterectomy.

I prefer to operate at, or near, full term so as to give the obstructing fibroid every chance of rising, for this has occurred as late as the second stage of labour.

In pre-antiseptic days such cases of Cæsarean hysterectomy were terribly fatal. Now the mortality is about 6 per cent. instead of at least 75 per cent. as in 1880.

I show you two specimens of uteri with fibroids which I removed at full term at this hospital, both mother and children surviving.

Case 4.—T. M—, a primigravida, æt. 33 years, married five years. Admitted to Charing Cross Hospital September, 1899. She complained of abdominal tumour, with three months' amenorrhœa. On abdominal examination there was a large fibroid tumour occupying the hypogastric, umbilical, and right iliac regions, and between the fibroids the uterine wall had an elastic feeling which suggested pregnancy. *Per vaginam* the cervix was out of reach, and the true pelvis was occupied by a large impacted fibroid. A provisional diagnosis of a three months' pregnancy in a fibroid uterus was made. Unsuccessful efforts were made by digital pressure under anæsthesia, and by hydrostatic pressure, to elevate the impacted fibroid. "Certain" signs of pregnancy were noted at the seventh month. The situation was explained to the patient, and as her desire to have a living child was great, and nothing was to be gained by operating then, it was decided to wait and perform a Cæsarean operation when near full term. On February 24th (thirty three and a half weeks of gestation) she had some severe abdominal pain and was re-admitted.

On examination *per vaginam* the large fibroid

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which had previously occupied the pelvis was found to have risen almost above the brim, and as the cervix was now in nearly its normal position it was thought that labour would be natural. Attempts to induce labour by introducing a bougie, by Steele's hydrostatic dilator, and by rupturing the membranes, whilst apparently causing labour "pains," failed to produce any sensation of pain, except when the hydrostatic dilator was *in situ* and the head did not descend into the pelvis. A well-marked caput succedaneum, however, was subsequently found, proving that the intermittent uterine contractions were true labour pains. As no real dilatation of the internal os took place, and as the fœtal head was felt high up above the *os internum*, resting on a bulging submucous fibroid, I performed Cæsarean section and removed the uterus below the lowest fibroid, treating the stump by the usual retroperitoneal method and leaving one ovary *in situ*. Owing to the position of the fibroids I had to incise the uterus through the posterior wall, coming on to the placenta, which I stripped off. The convalescence was uneventful.

Case 5.—Mrs. P—, a primigravida, æt. 34 years. Married eight years. Admitted to Charing Cross Hospital October, 1901. Patient was evidently pregnant, with a large abdominal fibroid passing down into the pelvis on the right side. The cervix was high up in front behind the pubes, and the whole of the sacral hollow was occupied by large nodular masses narrowing the obstetric conjugate to less than one inch. This retro-uterine mass seemed under A.C.E. anæsthesia to be quite fixed, and as Nature did not in this case elevate it from the pelvis, as in the last case, I performed Cæsarean section with Dr. Eden's assistance, and removed the uterus, as in the last case, ten days before labour was due. The retro-uterine tumour was found to be firmly adherent to the pelvic floor by vascular adhesions. Mother and child convalesced admirably.

You will see that in both cases the obstruction was absolute, in the one case by a submucous fibroid just above the internal os, in the other by a large fibroid adherent to the pelvic floor.

There are practically no alternatives to Cæsarean section (with or without hysterectomy) in these cases, for no attempt should be made to drag a living or a mutilated child past an obstructing fibroid by forceps or after version, so great is the danger to the patient. Statistics show that the mortality in these cases of delivery by forceps is from 25 to 60 per cent.; by version the mortality is 50 to 60 per cent., and after embryotomy about 80 per cent.

I have no time to discuss the possible alternative treatment of enucleation *per vaginam* where a cervical fibroid obstructs, but the risk of hæmorrhage and subsequent sepsis is great. Nor can I discuss the question of inducing sterilisation of the patient where Cæsarean section alone or myomectomy is performed, nor the question whether supra-vaginal hysterectomy or pan-hysterectomy is preferable. All such questions must be decided at the time.

II, Ovarian Cysts complicating Pregnancy are relatively rare.

Cysts are met with in all positions. I have seen them in the hollow of the sacrum, or lying side by side with the uterus in the abdomen, or obliquely across its fundus, or if small enough elevated and pushed backwards into one or other loin. Some of you saw me remove last week a cyst (Case 6) lying high up on the left side. Pregnancy was not four months advanced, but the uterus was at the level of the umbilicus owing to being perched up by a sub-peritoneal fibroid about the level of the reflection of the bladder anteriorly.

An elastic swelling was felt under the left ribs, extending backwards into the loin. This moved too readily for it to be a renal tumour, as it could be pushed into the left iliac fossa, and across, above the uterus, to the opposite hypochondriac region.

The patient had had an attack of pain and vomiting fourteen days previous to the operation, and we found, as suspected, that the pedicle was twisted. The cyst was dermoid—full of fat and hair.

It is remarkable how relatively frequent the cysts are dermoid when found complicating pregnancy. There is no sufficient explanation of this. The diagnosis of these cysts is usually easy enough if seen during pregnancy, though more difficult when actually obstructing labour.

Supposing a woman to have an ovarian cyst in the abdomen when about six months pregnant, the abdomen would be larger than normal. The uterus might be perched up on the cyst if it were detained in the pelvis, or the cyst might be lying side by side with it. Sometimes no definite line of demarcation can be at first felt, though occasionally fœtal movement or limbs can be felt over part of the tumour on one side, but not on the

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other. Abdominal ballottement, the fœtal heart, or the uterine souffle would also help differentiation.

The most reliable sign is, however, "intermittent uterine contractions," which, of course, are absent from the ovarian cyst. Such signs prove the case to be not one of hydramnios, for in that case the whole tumour would contract. If the cyst is in the pelvis, and especially if first discovered when the patient is in labour, the diagnosis has to be made chiefly from a fibroid, and it may not be possible to be certain, especially if the cyst be a dermoid.

Treatment.—It is bad practice to induce abortion or premature labour if an ovarian cyst is complicating pregnancy. That used to be an alternative, before abdominal section for an ovarian cyst became so safe. In such a case it is advisable to remove the cyst as soon as it is diagnosed. Otherwise it may get its pedicle twisted, when inflammation of the cyst and its surrounding peritoneum would follow, or there may be hæmorrhage into the cyst, which might then rupture. These accidents may happen at any time during the pregnancy or puerperium. If the cyst be abdominal, laparotomy should be performed, as in the case you saw me do last week. Premature labour does not often follow such an operation.

If the cyst be pelvic, and the patient be in labour, it is sometimes possible to push up the cyst and to deliver by forceps. It may also be possible to operate *per vaginam*, but unless the patient is actually in labour and the head completely engaged in the pelvis, laparotomy is far preferable.

Cases have been reported where, in order to overcome obstruction, the contents of the cyst have been removed by vaginal tapping or by incision into the cyst after posterior colpotomy, and the child delivered, leaving the cyst to be removed afterwards, either *per vaginam* or by abdominal section. Tapping would not empty a dermoid cyst, and post-colpotomy would then be necessary. The cyst may then be drawn through the vaginal opening, and if its pedicle can be reached, it should be ligatured, and the cyst cut away. If the pedicle cannot be then reached, deliver the child by forceps or version past the collapsed cyst, and then try to ligature the pedicle. Failing that, perform laparotomy afterwards

As an ovarian cyst is very liable to undergo torsion of its pedicle after labour, ovariotomy may be required during the puerperium. If, however, no such complication should occur, and the cyst was only discovered during labour and an immediate operation was not considered necessary, it is better to wait a month or six weeks, to allow the uterus to involute and the abdominal wall to recover its tone.

May 21st, 1906.

SERUM THERAPY.

A Clinical Debate held under the auspices of the Chelsea Clinical Society.

DR. BALL (President) in the Chair.

Professor R. T. Hewlett. Dr. Foord Caiger.
Dr. A. E. Wright. Dr. Allan Macfadyen.
Dr. Bertram Abrahams.

(*Concluded from p.* 80.)

Dr. Foord Caiger: Mr. President and Gentlemen,—I take the opportunity of expressing my pleasure in accepting the invitation of the Council to come and take part in the discussion of what is a most interesting subject. But all our joys are apt to be tempered with regrets, and I am conscious of the fact that I ought to have written a paper to offer to you to-night. But instead of that I am sorry I shall have to ask you to listen to a few remarks without any preparation in the form of a paper. It was a great shock to me to receive a letter from your Editorial Secretary asking me for a copy of my address. I quite misapprehended the proceedings on these evenings which are given up to debate, and I have to confess that, like the snakes in Iceland, there is no address. Therefore, I ask you with all humility to accept a few remarks of a desultory, though practical, character on a subject which is of undoubted interest.

Not having been present last week, I do not know the ground which was traversed by Dr. Wright and Dr. Hewlett. But as they are both eminent pathologists, I infer that their remarks were probably of a bacteriological and pathological nature. Therefore I propose to address myself entirely to the clinical aspect of the subject, and to practically confine my remarks to that branch of it of which I have had practical experience. The subject of serum therapeutics is one to which I have devoted much personal interest for the last ten or twelve years—in fact, ever since Roux read his historical paper at the International Medical Congress at Buda Pesth in the year 1894; and it must be obvious that to those who, like myself, have charge of an institution containing a large number of infective sick the subject of the treatment of infective diseases must be one of great importance I do not propose to weary you by

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reiterating in connection with diphtheria much statistical evidence. I think one may fairly say that the antitoxin treatment of diphtheria stands on a well-established basis. But I venture to remind you that in the hospitals of the Metropolitan Asylums Board during the last ten years some 60,000 or 70,000 cases of diphtheria have been treated, with the result that the mortality, which previously had never been below 30 per cent., has been reduced to less than 10 per cent. I have not the combined results for 1905, because they are not yet published, but I may say in relation to my own hospital that the mortality last year came out at only a trifle over 8 per cent. You will thus see what an enormous benefit has accrued in the public hospitals of London. And it is more particularly in connection with two classes of case that this benefit has been most apparent.

First of all, the laryngeal group—in other words, those which may be classed as "diphtheritic croup." Before the anti-toxin days we never got anything better than a recovery rate of 30 per cent. I believe I am quite correct in saying that for the eight years previous to the introduction of antitoxin the recovery rate averaged considerably less than 30 per cent. But now, owing to the universal use of anti-toxin, we are dissatisfied if we get a recovery rate much below 70 per cent. So you see the enormous improvement there has been in respect to that class of disease. Another group of cases in which the benefit has been most strikingly shown is in connection with post-scarlatinal attacks of diphtheria—in other words, patients who, during convalescence from an attack of scarlet fever, have developed diphtheria. Prior to the use of antitoxin post-scarlatinal diphtheria was justly regarded as the scourge of the scarlet fever wards, and a large part of the mortality was directly due to it. As a matter of fact, the mortality amongst post-scarlatinal diphtheria cases, taking all attacks, both laryngeal and faucial, worked out at over 50 per cent., and in the laryngeal group it was 80 per cent., and sometimes it even reached 90 per cent. Now, what has been the effect of the introduction of anti-toxin on these cases? Simply that, instead of 80 or 90 per cent., the mortality has been reduced to less than 1 per cent. The reason is that these patients being already under medical care at the onset of diphtheria, we are enabled to treat the disease at the very earliest moment that it appears. And that leads one to emphasise the extreme importance of early treatment. I do not think any better evidence on this point can be obtained than is shown in the statistical statement published in the 'Reports of the Metropolitan Asylums Board,' by Dr. MacCombie of the Brook Hospital, who shows the result of the use of antitoxin during the last eight years, arranged according to the day on which the serum treatment was commenced. This record deals with 5300 consecutive attacks, with the following results: Amongst the cases treated on the first day of the illness, numbering 200 to 300, the mortality was *nil*. When it was given on the second day of illness the mortality was 4·5 per cent. When given on the third day it was 11·9 per cent. On the fourth day it was 17·5, and when the antitoxin was not given until the fifth day, or later, it was 18·9 per cent. I do not think anything could be much more striking than that.

Now, a few words in respect to the clinical effects which follow the use of the serum. At the end of the first year's experience of its use the Medical Superintendents of the Metropolitan Asylums Board Hospitals conferred together and drew up a statement. We pointed out in that statement that there were certainly five effects which might be expected to follow the use of the remedy, and the experience of the subsequent ten years has served but to confirm that opinion in every particular. There is not one of us who signed that Report who is inclined to go back upon a single opinion which he then expressed. I may, perhaps, be allowed to briefly reiterate these effects. In the first place, there is noticed a rapid diminution of the faucial swelling and consequent distress; secondly, a rapid diminution, if not an entire cessation, of the offensive discharge from the nose, which everyone knows is such a distressing feature in a large proportion of severe attacks. The patient, owing to the blocking of the nasal passages, is unable to breathe through the nose, and his discomfort is proportionately great. Thirdly, there is a limitation of the spread, and an earlier separation of the membrane. In the faucial cases this can be directly observed and the results tabulated. And we have statistical evidence confirming it. In the laryngeal cases it is shown, not only in the great improvement in the results of tracheotomy, but also in the lessened proportion of the attacks in which either tracheotomy or intubation is called

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for. We find now that it is most exceptional for a case to develop laryngeal obstruction sufficient to call for operative relief if the signs of croup are not already present at the time of admission to hospital, whereas in the old days even if no sign of stridor could be detected on admission, treatment was quite impotent to obviate the necessity for either tracheotomy or intubation. That state of things is now quite altered, and the number of tracheotomies performed in a year is less than half what it used to be. The fourth point which we noted was a rapid improvement in the aspect and general well-being of the patients. The improvement in this respect was very striking to any observer who was faimliar with the diphtheria wards. Instead of the spectacle of a number of patients in great distress, with swollen necks and stuffed-up noses, fretful and crying, such cases are now quite the exception, and in the few one does come across the condition lasts for a comparatively short time. A fact which bore indirectly upon this was the depressing effect it had on the nurses in the diphtheria wards at that time. It was quite unusual for a nurse to care to stay very long in charge of one of the diphtheria wards because she found the work so depressing. But nowadays the diphtheria wards are perhaps the most popular in the hospital, a fact which is mainly owing to the change in the general aspect of the patients and the greatly reduced mortality.

The last of the point in the Report on which we laid stress was that in the cases which die life is prolonged to an extent unknown with any other form of treatment. The length of time which fatal cases now live is on the average more than three days longer than before they were treated with antitoxin. In cases which proved fatal it used to be the rule rather than the exception for the patient to die in the early faucial stage of the disease, from toxæmia, whereas now in the majority of cases death occurs at a later stage of the attack, from a form of cardiac degeneration leading to progressive syncope, which is often wrongly spoken of as "cardiac paralysis."

Now, with regard to the liability to complications. Under the antitoxic treatment we find that the percentage incidence has somewhat increased, and if one looks the matter in the face, it is, I think, only what one would expect. Take the occurrence of albuminuria, for instance. Whereas in 1894 the percentage was 24, in 1904 it was 31 per cent. But after all, even this slight increase is quite possibly only apparent. Since the introduction of antitoxin, so much more attention has been devoted to the registration of the various complications met with in diphtheria, that it is quite likely that the presence of albumen is not infrequently recorded which previously would have escaped attention. The injection of large doses of horse-serum, whether anti-toxic or not, will itself set up temporary albuminuria, but it is a condition which has no pathological significance. One is prepared, therefore, to find that there is a slight increase in the incidence of albuminuria.

The other complication which is so frequent after diphtheria is paralysis, and we find that an incidence formerly of 13 per cent. has increased to 15 per cent., a difference which is very slight. When looked at critically, it is found that the cases which come early under treatment for the most part escape paralysis, whereas those which come in in a late stage of the disease get paralysis almost as often as before antitoxin was employed. And another point is that nowadays many patients live to be affected with paralysis who would have died in the early stage of the disease under former methods of treatment. So it is only natural to expect that there would be some increase in the occurrence of paralysis if the treatment is efficacious. It is only fair to mention that a certain number of attacks seem to prove refractory to the anti-toxin treatment. I cannot explain the fact, but a small proportion of cases, perhaps about 2 per cent., do not show the improvement one would expect. I suppose the most reasonable theory is that the organism against which the horse has been immunised has been of a different strain from that which is acting in the human body. The same sort of difficulty, but to a far greater extent, is encountered in connection with anti-streptococcal serum.

We now come to the question of dosage. Our views on that matter have varied considerably at different times, and the practice has varied in the hands of different observers. I think it may be said that there are two ways of looking at the subject. One view as to the necessities of the case is that the antitoxin should be piled in in a large dose at the outset; on the other hand, it is believed by many that by spreading the dosage over a longer

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period more benefit results. When the treatment by antitoxin first came in we were in the habit of injecting some 300 to 400 units, followed by a dose of half that quantity on the following day. Subsequently it was found that the amount necessary was much larger, and we gradually increased the dosage, until we were in the habit of injecting 4000 units every few hours until the membrane showed clear evidence of separating. Often a patient got as much as an average of 1000 units per hour for two or three days, and it was not uncommon for 48,000 to 60,000 units to be given before the treatment was completed. Recently we have come to adopt a more modified view, namely that the larger initial doses are unnecessary, and that if we give a dose of 6000 to 8000 units, and repeat it at the end of twelve to twenty-four hours, according to the urgency of the case, and continue that treatment until the membrane shows definite signs of separating, we get as much benefit as from much larger doses. It is a point upon which I am often asked to express an opinion. Of course, the dosage should be regulated according to the amount of toxin which is assumed to be present in an unfixed condition in the system, and which, therefore, requires to be neutralised. According to the opinion recently expressed by Cobbett, it is only the toxin which has been formed in the previous half an hour or so which is unfixed, and which can, therefore, be attacked by antitoxin. And on that view this authority asserts his disbelief in the necessity of giving colossal doses of antitoxin at all. But after all it is very difficult to know what he means by the term "colossal." A thousand units is not colossal in one sense if it be given in the late stage of an attack, but it might certainly be regarded as colossal if given very early. I should determine my dose of antitoxin according to two factors: first of all the urgency of the symptoms, more particularly in the case of laryngeal attacks; and secondly, the time at which the patient comes under treatment—that is to say, the stage which the illness has reached when the treatment is commenced. There can be no question that 1000 units on the first day is far better than 3000 units on the third day, and so on.

Next, with regard to the method of administration. The usual custom is to give it subcutaneously. But if a more rapid effect is desired, owing to the urgency of the symptoms, this can be secured by adopting the method of intravenous injection. As a matter of fact, I have not personally had a very wide experience of intravenous injection. I am bound to say that in those cases where I have known it given in that way the attacks have been fatal. But, of course, it must be remembered that it was administered in desperate circumstances. The subcutaneous method is satisfactory in the majority of cases. It has been seriously recommended that the antitoxin should be given by the mouth, and even by the rectum. But I have never yet found sufficient, shall I say, courage, to trust to its administration in either of those ways. A few years ago a certain sportsman seriously recommended in one of the medical journals the giving of antitoxin by the rectum, and he quoted several cases which he believed to have justified the procedure, for they recovered. His treatment consisted in the introduction of a few hundred units *per rectum*, and the reason he gave for choosing that channel was that it did away with the difficulty of giving an hypodermic injection to a struggling child!

The complications due to the injection of antitoxin have a certain importance, although the tendency is to minimise them. We find that in something like 40 per cent. of cases a distinct complication arises as a result of the injection. It is in the form of either an urticarial or erythematous rash, attended usually with a sudden rise of temperature, and, perhaps, with the accompaniment of pain and tenderness in the joints, or it may be that the joints are affected without there being any attendant rash. But this latter event is comparatively unusual. Definite arthritis, in most cases attended with a rise of temperature for one to three days, is seen in about 5 per cent. of cases. An interesting point is that this "serum reaction," instead of appearing immediately, comes on after a definite interval, usually eight to ten days after the injection, and the fact that it has this latent period is suggestive of an analogy between it and an infective process which also has its period of incubation. I do not think anyone can say whether the reaction is dependent on any special factor in connection with the serum, or whether it is due to a special reactive capacity on the part of the individual, but possibly Dr. Macfadyen may be able to give us information on that subject. At any rate, it is a very distressing complication in certain cases.

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I should mention also that there is always a risk of subcutaneous abscess developing when anything approaching carelessness has been shown in respect to the injection. There is no doubt that with proper care this possibility can be reduced to a minimum. In the South-Western Hospital last year we did not have a single injection abscess, although the number of injections given was considerably over a thousand.

Next, I would like to say a few words on the question of giving prophylactic doses of antitoxin, I cannot help thinking that the practice is somewhat more popular than is to my mind desirable. I have seen many cases, especially in the adult, in which the injection of anti-toxin has given rise to really serious discomfort, to say the least of it. Although I have known children sleep through the injection and apparently suffer little or no disturbance from it, yet in grown-up people not only may urticarial eruptions develop eight or ten days subsequently, accompanied by most intense itching, so that the patient's life is one of great misery for a day or two afterwards, but not infrequently very acute tenderness and muscular stiffness is complained of at the site of injection, and for some distance around it, coming on within a short time of receiving the antitoxin. I have more than once quoted the case of a medical man who came into my hospital with diphtheria, and who received antitoxin. This gave him great joy at the time, and he at once felt more comfortable in consequence of the safeguard which he felt sure it would afford him. But that feeling, I regret to say, only lasted a short time, and he always regards the day or two following the injection as about the worst period of his life. He was certainly very much upset by it. This is not an isolated case. I think it is a mistake to hold prophylactic injections of antitoxin too cheap. I would not hesitate to give it at sight, or on suspicion, in the case of a young child, even if the child were only "a contact," but when you have to do with an adult, and that adult is under medical inspection, I think it is a mistake to give prophylactic injections. You can afford to hold your hand under such circumstances, and give it on the first symptom of illness.

Now with regard to scarlet fever. The attempts to treat scarlet fever by a curative serum are based on the well-accepted fact, I think, that the serious lesions in scarlet fever are usually due to a local infection with streptococci, culminating eventually in a streptococcal septicæmia. Acting on that supposition, many attempts have naturally been made to treat the disease by antistreptococcal serum. This, however, does not commit one to the belief that the specific organism of scarlet fever is a streptococcus, as is contended by Klein and Mervyn Gordon, though there is no doubt that streptococci may be always detected in the scarlatinal throat. Still, so it is with practically every other kind of inflamed throat, and the same thing is occasionally true of the blood. Streptococci can be recovered from the blood in most fatal cases of septic scarlet fever, and a streptococcal septicæmia is very common in connection with ordinary septic processes quite unconnected with a scarlatinal attack. So I do not think one is justified in expecting that a streptococcus is necessarily the specific cause of the disease. Of course, Klein and Gordon —Gordon having more particularly followed up Klein's work and extended his inquiry—believe that it is possible to differentiate a particular variety of streptococcus, the one originally described by Kurth under the name *Streptococcus conglomeratus*. They claim that it is the specific cause of scarlet fever. But I think one may justly say that, not only the evidence at present available is not conclusive, but that it is not at all certain that a streptococcus is the specific cause at all. My own feeling is that it is not unlikely that the specific cause of scarlet fever may be found to be a protozoon, and not a bacterium in the ordinary sense of the term. Mallory in America describes certain protozoa of two forms as being present in the protoplasm of the epithelial cells of the skin during the eruptive period of the disease, and also a little later, and he believes them to be specific. But he is candid enough to admit that it is only a suspicion on his part, and that he is at present unable to confirm it.

In connection with the treatment of scarlet fever by antistreptococcal serum, I think one must give Baginsky credit for having first tried it on an extensive scale, and he published a record of his treatment of several hundred cases, with a mortality of 14 per cent. He himself spoke very favourably of it. It depends very much on what the character of the attacks was in which he tried it. If he gave it in cases of all degrees of severity, just as they came, without any attempt at selection, one would

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naturally regard 14 per cent. as a very high mortality; but, on the other hand, if he reserved it for the septic group alone, as we are in the habit of doing in this country, 14 per cent. is a very low mortality. I am not at all clear as to what was Baginsky's qualification.

Of the various anti-sera prepared for the treatment of streptococcal infections, I have tried several, but I fear with but little success. One of them, however, which, owing to the kindness of Dr. Besredka, of the Pasteur Institute in Paris, who kept me supplied with it, I was enabled to try somewhat extensively, appeared to be of more value. This serum, which was prepared from streptococci derived exclusively from cases of scarlet fever, I tried on something like fifty cases of bad septic scarlet fever, and the mortality was 36 per cent. With regard to the Besredka serum, I gave it only to those cases which I thought would be more likely to die than to recover, and the mortality, as I have said, was 36 per cent. The most striking fact was that in proportion as the injection was given early the results were better. In seventeen of the cases the injection was given within the first four days of the illness, and the mortality was 23 per cent. In seventeen other cases it was given on the fifth, sixth, or seventh day, and the mortality was 35 per cent. In ten cases it was given on the eighth, ninth, or tenth day, and the mortality was 50 per cent. Those figures show that, as in the case of diphtheria and other diseases, early injection is a matter of paramount importance. I could not determine any special factor in the attack which might lead one to believe it would be successful, except that in cases where the temperature ran at a lower level the results were better. But there is a field here for much further experimental work. I will not detain you longer, except to mention that my personal experience of the treatment of typhoid fever by means of a curative serum is *nil*. I have tried, but without success, to obtain some of the serum with which Professor Chantemesse claims to have achieved such striking results in typhoid fever. The records, as published, are certainly most encouraging. As you probably know, Professor Chantemesse claims to have prepared a serum with which he has reduced the mortality of typhoid fever to less than 5 per cent., and that in a series of over 700 attacks. But as to the nature of the serum which he uses, and the actual method of its preparation, there is considerable mystery.

Dr. Bertram Abrahams: I have to thank you, Mr. President and gentlemen, for kindly inviting me to speak at this debate. I also, like Dr. Caiger, have to apologise for not having prepared an address. And as I did not hear the speeches which were made at the last meeting I naturally do not feel in a position either to traverse them or to try your patience by going over the ground which has been already covered in connection with the pathological aspects of the question. I propose, therefore, simply to discuss with you certain facts which have come within my notice in the course of eight years' experience in a large general hospital, and I think that even on the question of diphtheria what I have to say is of some little interest as a contrast in regard to conditions from what Dr. Caiger sees at the fever hospitals.

Diphtheria.—With us at a general hospital when we have a moderately bad case of diphtheria we do not keep it, but send it to a fever hospital. We have not any diphtheria wards and we only keep urgent cases, so that our mortality from the disease can obviously never come down to anything like the figure which Dr. Caiger gives in the institution of which he has charge. The difference in the mortality is to be accounted for in that way. But I remember when I was a clerk in the diphtheria wards at University College Hospital the mortality from that disease was something near 50 per cent. and that virtually every case on which tracheotomy was performed died. Two years afterwards I was in the same wards as House Physician, and in the meantime the antitoxin treatment had come in. Then we had a mortality of only 17 per cent. in ordinary cases, and very little more from the tracheotomised cases. At Westminster Hospital the present mortality from diphtheria has been, during the last six years, 15·6 per cent., and, as I have already said, that is considerably higher than it would be if we kept the less severe cases instead of sending them to a fever hospital. No doubt most of the cases which are marked "discharged unrelieved," meaning that they are sent to a fever hospital, get better at those institutions.

Tetanus.—I have had some slight experience with regard to this, having had opportunities of

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seeing the use of anti-tetanic serum in a few acute cases. The difficulties with anti-tetanic serum are numerous. In the first place, one does not know that a patient has tetanus until he has it very badly—that is to say, until his nervous system is involved. In the second place, whereas the tetanic infection apparently spreads up along the nerves and particularly the motor nerves, the injections, if given subcutaneously, spread along the ordinary lymphatic or vascular channels, and so it is considered to be necessary, unless you can, so to speak, predict an attack of tetanus and give the serum before symptoms appear, to inject it into the brain itself. And that is borne out by my very small experience of four acute cases. To three of them I gave a subcutaneous injection. Two of them died and one recovered. One I injected intra-cerebrally and he recovered. That, of course, is not anything like a sufficiently large experience from which to make any deductions, but still I am led, from that, to believe that the statements now made as to the method of administration were correct.

Enteric fever.—With regard to enteric fever, in that respect I am again in a rather opposite position to Dr. Caiger, because many cases of this disease come into a general hospital at a very early stage of the complaint, and without knowing what is the matter with them. But when patients enter a fever hospital with enteric fever, it is only when they are known to have the disease. So we who are connected with a large general hospital have a much better chance, if we are on the look out for enteric, of treating the case early than can be possible in a special hospital. Therefore it is not surprising to find that the mortality in my own experience during the last few years in a general hospital is very much less than anything that is possible, I think, under present conditions in a special fever hospital. During the last three years the mortality at Westminster Hospital from typhoid has been under 3·5 per cent., a result which is comparable with the result which Professor Chantemesse claims to have obtained as a result of the use of his special serum. We have been very much interested in the work which Professor Chantemesse has been doing with his serum. I have asked him about it myself, and he has told me it is a certain cure, but I have not been able to obtain any for my own use. One thing strikes the observer in seeing enteric fever abroad—in France and in Germany particularly—and that is that the mortality is as a rule much greater than it is in London. It may be that they are graver cases, or that they are not diagnosed in time, or there is something wrong about the research. The mortality according to the Paris statistics during this period has been 17 per cent., whereas in Chantemesse's own "bastion" it has been something over 3 per cent. But, as Dr. Caiger, who is so great an authority on these cases, says, nobody knows what the serum used by the Professor is. Next, Professor Chantemesse talks about causing cases to abort in one, two, or three days. One would like to be informed how he knows that. He does not give any statement which would enable us to get information on that point. To my mind, the great difference which has come about in the last few years, in my own personal experience it is true, with regard to the mortality from typhoid fever is due to the Widal reaction. Nowadays, the instant a case of typhoid is suspected the Widal reaction is tried, in other words people are constantly on the *qui vive*. We have not altered our treatment in any way, we have not even gone in for baths at Westminster Hospital—though I think we might with advantage do so—but we have had a continual alertness, and this reaction has brought about a very early recognition of the disease, and consequently an equally early treatment of it. As Dr. Caiger has already pointed out, the great point is to get a case early enough, and then you will in all probability pull it through.

Tuberculosis.—With regard to tubercle, I asked Dr. Harwood to come here to-night to explain these charts of his researches, but unfortunately he could not. I have here, and I have distributed copies among those present (see Statistics), statistics of the opsonin work which has been done by him in the Clinical Laboratory at Westminster Hospital during the last three months. The tabulated result does not appear to come to very much when it is summarised in this way, but it means from three hundred to four hundred estimations, and therefore it represents a good deal of work. And I may say it is all done by the same people, and therefore it may be taken to be trustworthy and consistent. And the conclusion which one comes to from it is that all one can say about opsonins is that the opsonic index is at best of some value from the point of view of prognosis. I mean that, other things being

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equal, a case with a high opsonic index is more likely to get well than one with a lower opsonic index. Many of the people who have been so very keen on the treatment of tubercle by means of sera and bacterial products have said that if you have a case with a high opsonic index and you give it tuberculin it will get better. But our statistics show that if you have a case with a high opsonic index and you do not give tuberculin it will still get better. That is to say that the opsonin test is excellent in prognosis, but it does not come in in reference to treatment. We found that in cases in which the opsonic index was taken before and after an operation for the removal of tuberculous material, such as bone, the opsonic index went up immensely as the result of the operation. So, on the whole, one is not inclined to think that the opsonic test is going to be of very great service in treatment. Moreover, Dr. Hebb reminds me that the recent experiments of Professor Calmette and others of his school have led them to be very much inclined to point out the disadvantages of tuberculin, on the lines of putting fresh tuberculous material into a patient or animal already tuberculous. That is not theoretically defensible, and in Calmette's laboratory it has been experimentally shown to be a hindrance rather than a help in regard to the successful treatment of the disease. So, on the whole, I should be inclined to sum up my experience of the serum-therapeutics of tuberculosis in this way: (1) That tuberculin is of great value in diagnosis. Of this there is no doubt; in veterinary work it is essential, and it is of use in many cases of alleged or doubtful abdominal tubercle. (2) That the opsonic index is of great value in prognosis.

Blood Examinations during three Months at Westminster Hospital for Estimation of Tuberculo-Opsonic Index, by Dr. C. S. Harwood.

Estimations made on twenty-one cases, mostly definitely tuberculous, some of doubtful diagnosis, and on a few healthy persons for comparison, as shown in appended table. The greater number of definitely tuberculous cases showed indices above the normal limit (0·8—1·2 taken as normal limits), possibly owing to several being phthisical, or to the index in other cases having been taken subsequent to operation. Of the doubtful cases none of those showing normal indices have as yet been proved tuberculous, but no opportunity has arisen so far of deciding the matter in the case of those in which the index indicated active tuberculosis. All the normal individuals whose blood was examined were inside the normal limits.

Tuberculin treatment was tried in four cases, as follows:

(1) G. D—, æt. 50 years. Tuberculosis of bladder (T.B. in urine); phthisis; pyrexia. Index, 0·6; four injections of $\frac{1}{800}$ milligramme each. Frequency and discomfort increased for a day or two after each injection, followed by slight amelioration. Negative phase long. As there was no marked improvement, injections stopped.

(2) F. G—, æt. 18 years. Multiple tuberculous lesions affecting bones of both feet, finger, elbow, and rib. Index 0·6. Operation: scraping, and one finger amputated. Index some days later 1·0. Then two injections of tuberculin, $\frac{1}{800}$ milligramme. Patient did very well, and was discharged after five weeks in hospital; not seen since.

(3) E. B—, æt. 18 years. Recurrent tuberculous keratitis three years. Enlarging mass of glands in abdomen; pyrexia. Index, 1·4, but fluctuating. Three injections of tuberculin, $\frac{1}{1400}$ milligramme, in four weeks. No obvious improvement, but still under treatment.

(4) W. W—, æt. 36 years. Tuberculosis of bladder (T.B. in urine); no pyrexia. Index 0·85. Injection $\frac{1}{2000}$ milligramme. Frequency and discomfort increased two days, with slight but well-marked rise of temperature. Frequency on fourth day decreased from 40 to 28. Still under treatment.

In all cases where sufficient examinations were made a negative phase was detected following injections, or in one or two cases a rise preceding the negative and another following the latter.

Dr. Harwood's Statistics.

(A) Four normal individuals showed indices, 0·87, 1, 0·9, 0·95.

(B) *Cases of undoubted tuberculosis.*—Ten.

	Under first examination.
Cyst in abdominal wall (T.B. in pus)	1·5*
Tuberculous peritonitis . . .	1·4
Phthisis with hæmoptysis . . .	1·5

* After operation.

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	Under first examination.
Caries of spine, psoas, and lumbar abscess	1·6
Phthisis (T.B. in sputum), advanced	1·6
Phthisis and laryngeal tuberculosis	1·4
Tuberculosis of bladder (T.B. in urine)	0·85
Tuberculous meningitis (T.B. in cerebro-spinal fluid)	1·3
Healed lupus of elbow	0·95
Tuberculosis of bladder (T.B. in urine)	0·6

(c) *Cases of doubtful diagnosis.*—Five.

*Tuberculosis (?) of bladder (no T.B. in many urine examinations; no reaction with Koch's old tuberculin for diagnosis)	0·95†
Malignant disease of lung and abdomen	1·1
Intra-cranial symptoms	0·85
Old caries of spine (?) residual or active	1·5
Lymphadenomatous glands of neck	2·0

Infective endocarditis.—The next point upon which I would like to say a few words is that of infective endocarditis, because it has been claimed that a great deal can be done for infective endocarditis by sera, particularly antistreptococcic sera. In the first place, one ought to say that it is impossible to lay down a pathological parallelism between the ulcerative endocarditis of the post-mortem table and the infective endocarditis which we diagnose during life. That is to say, I have seen cases in which the temperature has been subnormal throughout, and in which at the autopsy there were multiple abscesses and ulcers on the valves. I have seen other cases in which there has been a hectic temperature for weeks, but in which at death no suppuration was to be detected. One's great test must be an examination of the blood, and it has long been a routine practice with me to examine the blood in all cases in which there is a reasonable suspicion of micro-organisms being found. The result of that is not, on the whole, as encouraging as one would hope, because one finds that these infective processes are due to gonococci and other organisms as well as to streptococci, therefore one does not find that cure of all cases which at one time was held in prospect. Certainly I have seen cases get better under antistreptococcic serum, in which cases streptococci have been found in the blood; and I have at the present moment a patient, or a young lady who was a patient, who comes to see me once a year or so, who had infective endocarditis and whose blood contained streptococci. She had injections of a mixed serum—which, of course, is adopting a rather blunderbuss method of treatment—because one did not know what strains of bacilli were causing the illness. But, at any rate, she recovered, and, as evidence of the fact that she had endocarditis, she has, to this day, a pulmonary regurgitant murmur, the only one I have heard. It is not, however, excessively rare, because the late Dr. Washbourne told me that he had had a case too. So in infective endocarditis there is some chance of your curing your patient by a serum if you find streptococci in the blood and if you can give the right strain. Otherwise I am afraid the hopes from serum-therapy in that disease are very limited. On the other hand, in a well-known streptococcic infection such as that of erysipelas the results are often most striking and remarkable. Of course, my experience in this direction is limited, but now and then a physician sees a case of that disease, and there, almost invariably, anti-streptococcic serum is effectual.

Acute rheumatism.—Now a word on rheumatism. It has been averred many times, but always by the same people, that rheumatism is due to a specific diplococcus. This diplococcus has had a long and chequered history. It was originally discovered by Triboulet and subsequently discarded by him. Then it was re-discovered by Wassermann and discarded by him, as the cause of rheumatism, that is to say, but before the Aliens Act was passed it came over to England and was received with open arms. But the belief in this particular diplococcus as the cause of rheumatism is limited to one or two people who are apparently not such great believers in Koch's laws as many others are. There are a large number of pathologists in London who have never been able to obtain it from rheumatic lesions. Where the question touches us in this debate is that one of the devotees of this particular diplococcus—Menzer—made an anti-diplococcus

* This patient has since died of tuberculous meningitis. T.B. were found in the cerebro-spinal fluid during life. Tuberculosis of the bladder was discovered at the post-mortem examination.—B. A.

† Hardly varying in four examinations.

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serum, and he wrote a book on the subject, and brought his results forward at the Berlin Clinical Society. There the whole thing was very much discredited, and nothing has been heard of it since. That happened three or four years ago. I do not think the serum treatment of rheumatism is making any progress at present.

Exophthalmic goitre.—I have not had any experience of the serum treatment of exophthalmic goitre. That is a disease which in ordinary practical treatment you can do nothing for. I know of no remedy which does it the slightest good, except rest in bed. I have myself tried belladonna and various other drugs, and have tried, usually in vain, to persuade myself that they are of use. I am certain that no operation which has been devised has done good. Therefore I think serum treatment, the serum having been obtained from a goat which has been deprived of its thyroid, might do good in such cases, and I intend to try it.

Lastly, I would say a word about the use of diphtheria serum in other conditions, because every now and then you find people who tell you that they have treated cases of different kinds with anti-diphtheria serum, and with benefit. This has, for instance, been alleged with regard to phthisis. Cases have been recorded in which anti-diphtheritic serum has apparently produced a temporary alleviation in cases of phthisis, and Dr. Blanc, of Aix-les-Bains, who is a well-known authority on rheumatoid arthritis, tells me he has had good results in some of his bad cases by giving injections of anti-diphtheritic serum. There is no doubt that anti-diphtheritic serum causes a certain amount of phagocytosis, and therefore that it might possibly alleviate any condition in which the leucocytes were getting the worst of the struggle. Beyond that I cannot see what scientific grounds there are for its use, and I do not think it is to be recommended in any of these cases, as long as we have other and better means of inducing phagocytosis at our command.

Dr. ALLAN MACFADYEN said: I am not in a position to speak on the subject of serum therapeutics from the clinical standpoint, as my work lies in the experimental field. My researches have been devoted in large part to the investigation of bacterial toxins, because I believe that the future progress of serum therapy will largely depend on the fresh knowledge that may be gained with regard to the toxins produced by infective micro-organisms. Of these toxins a great deal still remains to be learned, and I have no doubt that research will in time make valuable additions to our knowledge.

The aims of sero-therapy are twofold, viz. to effect an immunisation against the morbid agent or its products. Serums are accordingly of two classes, the anti-bacterial and the anti-toxic. The anti-diphtheria and tetanus serums are the classical examples of the latter group. In the case of diphtheria and tetanus the specific agents secrete a poison at the seat of their invasion, to which the grave symptoms of these diseases are due. One has, therefore, to combat not so much the local infection as the general intoxication resulting from the infection. [A description was given of the experimental methods which had led to the successful preparation of the diphtheria and tetanus serums. These serums were anti-toxic, not anti-bacterial, and their use was based on a rational system of therapeutics in the case of the diseases referred to.]

The observations upon diphtheria and tetanus naturally led to investigations with regard to the production of soluble poisons by other pathogenic bacteria. The results, however, were in the main negative or disappointing. The typhoid and cholera organisms may be taken as typical examples. Filtered fluid cultures of these organisms proved to be non-toxic or only feebly toxic. It was found that the body-substance of the bacteria was much more toxic than the media in which they had been cultivated, in contra-distinction to diphtheria and tetanus. This led to the recognition of a group of bacterial poisons, termed "intra-cellular," inasmuch as they are not secreted like the diphtheria toxin, and are only liberated on the disintegration or solution of the cell. The demonstrable toxins of a number of infective bacteria are of this intra-cellular type. It has generally been deemed impossible to produce antitoxins for poisons of this character. Attention has, therefore, been almost entirely devoted to the preparation of anti-bacterial serums with the aid of dead and living cultures of the micro-organisms. Of these the anti-streptococcic serum is a familiar example. As previous speakers have indicated, their success as curative agents is a matter of considerable doubt. A fresh difficulty was that a serum, although potent against

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the particular strain used for immunisation, might fail in the presence of other strains of streptococci. This led to the preparation of polyvalent serums with streptococci isolated from as many sources as possible. These serums are still on their trial, and opinion is divided as to their value. It must be acknowledged that the anti-bacterial have not the established therapeutic value of the anti-toxic serums.

Reference has been made to toxins of an intra-cellular type, as in the case of typhoid and cholera. To these may be added the toxins of the plague bacillus and other members of the hæmorrhagic septicæmic group.

Chantemesse prepared an anti-typhoid serum, using a toxin obtained by cultivation of the bacilli in a special and complicated medium. The serum is anti-toxic and Chantemesse has found it to be of value in enteric fever. His results are certainly an encouragement to further experimental work in this direction. It may be that the toxin obtained by Chantemesse is really of an intra-cellular type, and has been liberated in the medium employed by disintegration of the typhoid bacilli. The same may be said of the cholera toxin described by Metchnikoff and used by him for immunising purposes. The serum obtained was only feebly anti-toxic and has not, so far as is known, been put to the clinical test.

Research had meanwhile failed in the attempt to produce efficient serums for toxins of a strictly intra-cellular type; and one of the great difficulties lay in the extreme sensitiveness of experimental animals to poisons of this character. In the case of the dysentery bacillus an intra-cellular toxin can be soaked out in an alkaline culture medium and an anti-toxic dysentery serum prepared. Such a method cannot, however, be applied to cholera, typhoid, or plague, nor can one employ chemical methods of extraction which alter or destroy the toxins. Buchner and Hahn, by crushing bacteria with sand, and Kieselguhr obtained the cell-plasma of pathogenic organisms—*e. g.* typho-plasmin. The serums prepared with such extracts were anti-bacterial, but not antitoxic. Autolysis has also been employed, but there is no record of serums successfully prepared by the aid of this method.

[Reference was made to the cold-grinding methods employed by the speaker for the purpose of extracting these intra-cellular toxins. A description was given of the immunising experiments that had been carried out on the goat, whereby it had been found possible to produce a serum of marked anti-toxic power. At the end of four months' immunisation $\frac{1}{50}$ c.c. of the goat's serum neutralised thirty lethal doses of the typhoid toxin. A very marked rise in the antitoxic value of the serum had therefore been brought about after a comparatively short period of immunisation. Analogous results have likewise been obtained in the case of the cholera toxin. It has now been amply demonstrated that it is possible to produce in considerable amount anti-bodies for this important class of toxins. This is an important step in advance. The value of such a serum in the treatment of the clinical disease remains to be tested. The next step will be to ascertain how far it is feasible to induce in the horse an equivalent rise in the antitoxic value of its serum.]

With regard to serum therapeutics bacteriology must concern itself more diligently with the study of bacterial toxins of this nature and ascertain in how far an anti-toxic element can be introduced into the serum treatment of such diseases as typhoid, cholera, and plague. Advances have already been made in this difficult and promising line of inquiry.

We have received from Burroughs Wellcome & Co. samples of their Tabloid "Xaxa." This is a trade mark name to designate acetyl-salicylic acid issued by that firm. Acetyl-salicylic acid has been widely prescribed, as it appears to possess all the valuable therapeutic properties of salicylic acid and its salts, without unpleasant after-effects. Acetyl-salicylic acid is insoluble in the stomach, but readily soluble in the intestine, and therefore causes no gastric irritation. It may be used in any conditions in which salicylic acid or sodium salicylate is usually employed. It is unquestionably of great service in gout and rheumatism especially in connection with the ear affections associated with these conditions. It is also of conspicuous value in some forms of headache and neuralgia. For the relief of pain, fifteen grains may be prescribed as a first dose, with further doses, if necessary, of ten grains at intervals of one hour to one hour and a half, until three or four doses in all have been taken.

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ABSCESSES IN CONNECTION WITH SPINAL CARIES AND THEIR TREATMENT.

A Lecture delivered at the Hospital for Sick Children, Great Ormond Street.

By T. H. KELLOCK, M.A., F.R.C.S.,
Surgeon to the Hospital.

LADIES AND GENTLEMEN,—The title I have given to the subject for our consideration to-day was intended to show that I do not propose to deal with the symptoms or treatment of spinal caries itself, but rather to call your attention to one of its complications, and, as far as our time will permit, to discuss the pathology and treatment of the so-called abscesses which arise in connection with it. I do not suppose that in the whole realm of surgery (for the condition is one that is by no means confined to children) there is a province in which greater change for the better has taken place during the last ten or fifteen years than in the treatment of these abscesses. To the casual observer this may not be so very evident, but to those who are constantly associated with this affection the improvement in the prognosis which has been brought about is very great. About the period I have mentioned it was by no means uncommon to find a number of the surgical beds in this hospital occupied by children suffering from tuberculous disease of the spine, bones, and joints, in whom suppuration had been added to the tuberculous affection, sinuses had formed, at the bottom of which septic tuberculous osteitis was to be found; the patients were ill, thin and wasted; pain, hectic fever, and occasionally lardaceous disease of viscera were their lot; general disseminated tuberculosis was not uncommon, and the outlook, even if they escaped with their lives, was a tedious and prolonged convalescence. Now, I am happy to say, matters are much better; I will not say that even

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now such cases are very exceptional, but the number of those in which primary union of the wounds made for the evacuation of the abscesses and the recovery of the patients from this complication, with the maintenance of a good or fair condition of general health throughout, is enormously increased. The reason of this improved condition of things is not very far to seek; it is based, I think, on the better appreciation of the fact that in this connection the terms "abscess" and "pus" are wrong and misleading. It is true they are convenient terms and sanctioned by long use, but these are neither abscesses nor pus according to the usual acceptance of the terms; they are collections of broken-down tuberculous granulation-tissue, there is nothing septic about them, and the interior of an "abscess" in connection with tuberculous caries of the spine may be as free from sepsis and as surgically clean as a normal peritoneum or a healthy joint; the material found in these abscesses is pathogenic in the sense that it is tuberculous; for although it is almost impossible, or at any rate very difficult, to demonstrate in it the presence of tubercle bacilli, yet we know that it is capable of setting up a very definite tuberculosis if injected into the healthy peritoneal cavity of an animal—a fact which should make us particularly careful when dealing with collections of this material lying in close contact with the peritoneum, as in the case of an iliac abscess.

If, then, in the course of what I have to say to-day I use for brevity's sake the terms "pus" and "abscess," I must ask you to accept them as meaning a collection of broken-down tuberculous material and the cavity which contains it respectively, and not in the sense that they are of the septic nature that these terms generally imply.

The manner in which the abscesses form and increase in size is of some importance; it is more than likely that only a proportion of the pus comes from the actual carious spot or spots in the vertebræ. What happens, I think, is that the carious spot gives rise to a small collection of pus in its immediate neighbourhood, the surrounding tissues become infected, and from the walls of the cavity of this abscess, lined as it is by tuberculous granulation-tissue, comes by far the greater part of the pus; the recognition of this fact has an important bearing when we come to consider the treatment of these collections, for it would be almost useless to deal with the abscess cavity and its contents alone if the diseased bone were the sole source from which the pus were derived. Another important point to which I should like here to call your attention is the comparative harmlessness of these abscesses in themselves to the patient's general health; a child will go on for months, or even years, with an abscess in connection with caries of the spine and be little the worse for it; in fact, he may be unaware of its presence, and it may be that it is only discovered by accident; it is when, by some means or other, septic infection is superadded that danger arises, and often I think it is better for the patient to be left with the abscess intact until his general health has been attended to, rather than that the abscess should be opened and suppuration allowed to get to the seat of the disease.

A question which is sometimes put to one, "Is there always an abscess in connection with tuberculous caries of the spine?" should, I think, be generally answered in the affirmative. One rarely meets with tuberculous disease of bones, especially of soft cancellous bones like those of the bodies of the vertebræ, without finding a certain amount of broken-down material around. It may be, and often is, that this collection is small, but that is only a matter of degree, of the extent of bone affected, and of the length of time during which the disease has been active. What, then, happens in those cases that get well without the formation of any abscess that can be detected? or, in other words, does an abscess in connection with spinal caries ever get well by itself? Here, again, I think the answer should be Yes. I have known this happen in a child whom I had under observation for a long time; she had caries of the dorsal spine, and, at one time, a distinctly palpable psoas abscess. This was not operated upon on account of the child's general condition, and, in the course of time, it appeared to solidify, and she made a complete recovery as far as the abscess was concerned. Further, let me remind you of what happens in cases of spinal disease complicated by paraplegia. You know that the operation of laminectomy in such cases was practised more frequently some years ago than it is at the present time, and during a rather long period of residence in this hospital I had the opportunity of seeing the results from the two forms of treatment, operative, and

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that by rest alone. Some of the surgeons would perform laminectomy, others would not; the latter would say, "Leave the abscess alone and take equally good care of the children as if the operation had been performed, and the prognosis will be equally good," and I think this was true; in both, with prolonged rest and attention to the general health, the prognosis was fairly good. What, then, became of the abscesses in the canal of the spinal column of those cases where no operation was performed? In all probability they became solidified or absorbed. We know that this occurs in tuberculous disease of the knee-joint and in tuberculous subcutaneous abscess, and why not therefore in connection with spinal abscesses as well?

The rate at which these abscesses develop forms a valuable guide as to prognosis, and this rate varies considerably; one sometimes sees children in whom a few months, or even weeks, after the first evidence of spinal disease an abscess of considerable size is already present, whilst, on the other hand, one may watch a case for months, or even years, and then casually make the discovery that an abscess has developed. What it is that determines the pace at which these collections form is a little difficult to say. I think the explanation is that, in the more rapidly developing ones, the disease is very active and extensive in the spine itself, and in those which accumulate more slowly that the disease in the bone is more or less quiescent, and the pus is forming almost entirely from the walls of a previously unnoticeable abscess around the original seat of the disease; anyhow, the prognosis after operation is very much better in the latter class.

No additional symptoms, as a rule, arise when a collection of pus originating in spinal caries becomes large enough to be detected on examination; on the contrary, there is sometimes an amelioration of some of the most serious of the symptoms—namely, those due to pressure on the spinal cord. It is quite exceptional to meet with a case where a patient suffers from paraplegia or other sign of pressure on the cord at the same time as he has a definite abscess which can be felt at a little distance from the spine, as, for example, in the iliac fossa, and the development of such an abscess where there have been signs of pressure on the cord is generally an indication that such signs will in all probability shortly disappear; the pressure finding vent, so to speak, externally ceases to exercise its injurious effect on the spinal cord. In this connection, however, one word of warning must be added: we know that paraplegia and loss of sensation are often the result of myelitis caused by direct extension of the tuberculous disease to the cord, and in such improvement when the pressure finds a vent must necessarily be much more delayed than when the symptoms are the result of pressure alone.

The direction in which these abscesses travel after once they are formed depends, apparently, in the first place, on the part of the body of the vertebra first affected by the disease: they come forwards, laterally, or backwards into the spinal canal, according as to whether their origin is in the front, side, or back of the body of the vertebra the first-named, of course, being by far the most common. When once they have, as it were, started on their journey they are easily guided by comparatively weak structures, such as fasciæ and sheaths of muscles. A good example of this is seen in the case of a psoas abscess; the pus finds its way into the muscle at the origin of the latter from the vertebra, the abscess may attain quite a considerable size, and yet such a thin covering as the sheath of this muscle is capable of keeping it within bounds. The same is seen where an abscess travels for a long distance in intermuscular planes without affecting or encroaching upon the muscles themselves. Unless sepsis has been added to the tuberculous affection these abscesses very rarely burst into neighbouring cavities; as a cause of empyema or tuberculous peritonitis the bursting into the pleura or peritoneum of a pure tuberculous abscess is a rare event. I have only once, I think, known an iliac abscess burst into the peritoneum. The patient was a little girl in this hospital; she had been brought up from the country with spinal caries and a rather large iliac abscess; the following morning she was found dead in bed, having apparently died whilst asleep; a post mortem showed that the abscess had burst into the peritoneum, and the shock of this must have caused her death. Should, however, sepsis be added to the tubercle, the abscesses will transgress all natural bounds and open into almost any cavity. I recently had under my care a man suffering from an iliac abscess which had in some

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way become infected with sepsis before any operation had been performed; the abscess was painful and tender, and his temperature of the hectic type; the abscess burst suddenly into the bladder, having first presumbly found its way through the iliac fascia and so into the pelvis. An abscess in connection with the lateral aspect of one of the dorsal vertebræ is more likely to find its way round the chest wall, guided by the planes of intercostal muscles, and come to the surface anteriorly where these planes are weak or deficent than it is to cause empyema by bursting into the pleura. In considering the situations in which these abscesses form, and taking the spine from above downwards, we find at the outset a fairly common and one of the most serious localities, the upper cervical region. Tuberculous caries is fairly common here, and an abscess in connection with it almost always tends to come forwards at first, and to cause the condition generally known as chronic retropharyngeal abscess. They very rarely burst into the mouth unless by the surgeon's finger or other means some violence is applied to them; they are more easily directed by the prevertebral fascia towards the side of the neck, generally the left, and making their way between the muscles, are often to be found forming a swelling immediately behind the upper part of the sternomastoid muscle. A chronic retropharyngeal abscess causes by itself few symptoms if it be not very large; a little difficulty in breathing or swallowing, or a little alteration in the voice may be all; but their early recognition is a matter of serious importance, indicating, as they do, caries of the spine in an extremely dangerous region. And not only is their recognition important, but so, also, is their differential diagnosis from other conditions in this situation. The acute retropharyngeal abscess of infants and tuberculous affection of the lymphoid tissue in this position may both closely simulate them, but these conditions, arising as they do in front of the prevertebral fascia, have quite a different origin, and call for quite different treatment to that due to caries of the spine. Were it not that experience says otherwise, it would seem unnecessary to say that care should be taken not to mistake an early retropharyngeal abscess for adenoid vegetations; that this mistake, with its serious consequences, may occur should always make one very careful in the digital examination of the back of the pharynx in cases of known or suspected swelling there, and shows the desirability of inspecting the parts before such an examination, for in cases of abscess in connection with spinal caries it generally happens that a part, at any rate, of the swelling can be actually seen a little farther down the spine. The lower cervical and upper dorsal region must detain us a little while, for abscesses arising in connection with caries here cause symptoms of the greatest importance; they are at first situated behind the upper opening of the thorax, an opening which in children is peculiarly small, and the result of pressure on the important structures passing through it may be very serious. In this situation the abscess generally comes forward, and flattening or pushing on one side the œsophagus, may exercise pressure on the trachea, when the latter is caught between the abscess behind and the upper part of the sternum in front. There are two rather different ways in which the results of this pressure show themselves; in the one the pressure may be sufficient to actually flatten out the trachea and so cause an obstruction to respiration which, if not relieved, may end fatally. I have seen, post mortem, a case where this had occurred, and where the lumen of the trachea had been reduced to a mere transverse slit. On the other hand, the pressure of the abscess may be slight, and the interference with respiration may then very closely resemble that which is sometimes seen in cases of aneurism in an adult. The dyspnœa may be spasmodic, and may possibly be due to pressure on the recurrent laryngeal nerves, causing thereby spasmodic contraction of the intra-laryngeal muscles; this spasmodic dyspnœa is generally most marked at night, and I have seen a child under these conditions happy, and even lively, during the greater part of the day but suffering at night from severe attacks of dyspnœa; they came on apparently directly she fell asleep, woke her, and then for an hour or two she would be seriously distressed. A more serious one than usual one night induced the performance of tracheotomy and the passage of a tube down the trachea to apparently below the obstruction; the child, however, died, and post-mortem examination showed an abscess containing no more than two drachms of pus which was in contact with, but not really narrowing, the trachea. A very similar condition of spasmodic dyspnœa is sometimes met with in cases of enlarged glands at

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the bifurcation of the trachea, but, of course, other evidence of caries of the lower cervical or upper dorsal vertebræ would be of very great importance in distinguishing between the two conditions. In cases of spasmodic dyspnœa in a child I would say, never omit a careful examination of the spine, for a very slight rigidity or irregularity may be the only palpable evidence of caries going on there sufficient to produce an abscess which may be the cause of the symptom. I do not think I need occupy your time by describing the courses taken by these abscesses when they originate in the middle and lower dorsal and the lumbar and sacral regions of the spine. You have probably all seen instances of these finding their way to the front of the chest, tracking along the intercostal spaces, showing themselves as lumbar collections, determined by slightly resistant structures into the pelvis, and coming to the surface in the sciatic or ischio-rectal regions. These are all more or less exceptional; the common course, that of the one we are most often called upon to diagnose and treat, is when the abscess, finding its way into the sheath of the psoas muscle where this takes its origin from the bodies of the vertebræ, remains confined within the limits of this sheath, till, reaching the iliac fossa, it transgresses the very slight barrier between the psoas muscle and the iliacus, and filling up the sheath of the latter as well, forms that so well known as ilio-psoas. Such collections will often remain thus for a considerable time, and will often attain a large size without going further. But if they are untreated it is under Poupart's ligament along the conjoined tendons of these muscles that they are found to advance and, leaving the tendon sheath at the lesser trochanter of the femur, will come forwards and present at the saphenous opening, an occurrence which will sometimes take place quite suddenly and cause the question of their distinction from a femoral hernia to arise. For, in common with the latter, their first appearance sometimes follows a strain or injury; at first, at any rate, the swelling is capable of reduction when the patient is lying down, and a distinct and expansile impulse on coughing is generally present. Progress of these ilio-psoas collections in other directions after they have entered the thigh are not by any means unknown, but the course which I have described is by far the most common.

We are brought now to the second part of our subject, namely the treatment of these abscesses, and as the same principles are applicable to them in nearly all situations, I would like to speak of it generally before mentioning a few points which appear important in the different situations. One general principle should underlie the treatment of them all: they should be dealt with as collections of tuberculous material, and not as septic abscesses, and, as I said before, it has been the general recognition of this which has brought about such a marked improvement in the prognosis in these cases. We can hardly hope by operation to cure the disease in the spine; we cannot excise the affected part of the column any more than we can hope to remove all the affected bone by scraping or gouging; but what we can do is to remove the products of this diseased bone, and also the lining of these cavities, which is the source of the greater part of their contents. The overcoming of the disease in the spine itself has to be left to natural means; and it goes without saying that throughout the surgical treatment of these cases everything that is appropriate should be done for the patient's general health, so that he may be the better able to gain the mastery over an affection of which even the disease in the bodies of the vertebra may be only a part.

As you will remember, I mentioned previously that in cases where the patient's general condition is not good and seems capable of improvement it is sometimes wise to delay operation for a time for fear of a want of power of repair in the wound, failure of which will almost certainly lead to a tuberculous, if not a septic, sinus leading down to the affected bone. Given a patient in a fit condition to have the operation performed, the following should be the general principles guiding one: These abscesses should be opened early—that is, while they are deeply situated; they should be opened through the thickest layer of healthy tissue available; they should *not* be opened in places where the skin is implicated, or in the most prominent and inviting positions where they are nearest to the surface. The reason for this is obvious; one wants the largest amount possible of unaffected tissue to bring together afterwards and form a barrier between the interior of the cavity and the surface of the body; for the same reason the openings should be made through, rather than between, muscles; for the subsequent closing of

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muscular structures divided in their long axes is more satisfactory than if the individual muscles are merely separated one from the other. After the abscess has been opened by deliberately dividing, layer by layer, the overlying structures, its contents are completely evacuated, the granulation tissue lining the walls gently scraped away, the cavity irrigated, and then thoroughly dried. Closure should be done with care; the structures that were divided last should be first accurately sewn, then the other coverings in the reverse order to that in which they were divided, and, finally, the skin wound closed and sealed. I think the solution used for the irrigation is a matter of little importance, for none of it is left in the cavity, and its action is almost, if not quite, a mechanical one; a weak solution of perchloride of mercury or plain sterilised water is what I generally employ. It has been rather a debated point whether anything should be left in these cavities before they are closed; various substances have been used, and used with rather different objects in view. An emulsion of iodoform in glycerine was once very popular, used with the idea that the iodoform would slowly disintegrate, and in doing so destroy any tubercle bacilli that were present; this disintegrating process often took a very long time, and it must have fallen to the lot of everyone who has had charge of children in convalescent homes to have to remove masses of iodoform from deep sinuses in cases of spinal caries. This iodoform emulsion may have been of some use; but while it remains it is of the nature of a foreign body, and as such prevents the rapid coalescence of the walls of the cavity, which is so much to be desired, and is, I think, very little used at the present time.

Small quantities of strong solution of zinc or carbolic acid are, or have been, frequently used with the idea that they will at once destroy any tubercle bacilli that may be exposed in the walls of the cavity; they may do so, but their action is very superficial, and their use is apt to be followed by a reactionary discharge of fluid into the cavity, and in this way prevent the coalescence of the walls. Recently in a few cases I left some solution of iodide of potassium in these cavities with the idea that it might be slowly absorbed by the walls, and during its absorption have some effect on tubercle bacilli in the surrounding structures, but in this I was disappointed. The walls of these cavities seem to have little power of absorption, and the fluid remained and had to be drawn off again in several instances. Another substance which is now used a good deal, I believe, but with quite a different object in view, is the solution of adrenalin. Should there be any difficulty in rendering the interior of the cavity quite dry a small quantity of this solution will often aid very considerably, and for this purpose I have found it very useful. One would not, of course, leave in the cavity any quantity sufficient to keep the walls apart, and, as far as my experience of it has gone, its use is not often followed by any reactionary discharge of fluid from the walls.

The objections to the employment of drainage of these cavities are very great. Prolonged drainage is very little used now; it generally means septic infection at some time or another, guard against it as carefully as we may; and even if it does not lead to septic infection it means the establishment of a sinus which will go on discharging thin tuberculous fluid for months, if not for years, to the marked disadvantage of the patient. Temporary drainage is another matter, and many surgeons are in the habit of employing it for twenty-four or thirty-six hours for the escape of the fluid which is apt to collect in the cavity immediately after the operation. This it does; but the objection to the practice is that the presence of a drain even for so short a time means that the opening in the walls of the cavity cannot be completely closed, and the removal of the drain must mean a weak spot in the operation wound, which is very likely to become infected with tubercle, and so eventually lead to the formation of a sinus. It is far better, I think, to use what means we have, such as swabbing out with adrenalin solution before closing, to prevent the effusion of this fluid, or if it does collect soon after the operation, remove it by means of a fine aspirating needle passed into the cavity from some spot on the surface other than that involved in the wound; for the fluid in this case is thin, and can be quite easily removed in this way, and if fluid re-collects in the cavity we like it to do so in the original situation, and to be completely shut in by the structures which originally enclosed the abscess. In the dressing of these cases the question sometimes arises whether we should apply pressure to keep the walls in contact and so aid coalescence.

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In the majority of cases this is, I think, quite unnecessary, and in the case of an ilio-psoas collection may even be harmful by exerting pressure on other parts, such as the intestine, at the same time. When an ilio-psoas abscess is first opened, and before the contents have escaped, if a finger be passed into the cavity this feels widely open, and it is often difficult to reach its limits; but when the contents have been evacuated the natural elasticity of the surrounding parts, aided by the respiratory movements, are generally sufficient to bring the walls in contact, and the cavity as such ceases to exist, reminding one very much of what happens in dealing with an empyema when the lung readily expands after the fluid has been set free. Should re-collection of fluid recur in the cavity within a day or two of the operation, it may be dealt with, as I have already mentioned, by aspiration, and this may if necessary be repeated; but after an interval of a week or more the fluid will probably partake more of the nature of the original contents, and the operation should be repeated. It should be done on the second occasion, if possible, through a fresh incision rather than through the scar of the former one; the latter would probably lead to a weakening and ultimate breaking down of the scar. Should it be absolutely necessary to do the second operation at the original site, the scar should be excised fairly widely, and the excision carried through all the layers that cover in the abscess. Occasionally one has to repeat the operation three or four times, but as a rule the cavity is found smaller and the contents less on each occasion, and eventually the fluid ceases to re-collect; in a very considerable proportion of the cases, however, one operation is sufficient, provided, of course, proper care is taken to keep the patient at rest so as to give the disease in the spine the best chance of becoming quiescent.

There are just one or two points in the application of the principles of the operation to specific regions of the body which are worth consideration. In dealing with the common ilio-psoas variety it is, I think, a general custom now to expose the abscess by splitting the muscles in the direction of their fibres rather than by dividing them transversely. It is true that as the incision is generally made fairly close to Poupart's ligament and the crest of the ilium, there is little danger of a subsequent hernia into the scar, and that more room is obtained by a transverse section of the muscles; yet if one's object is, as it should be, to obtain a firm and resistant barrier over the cavity, this is better attained by the splitting process. Having made such a dissection down to the peritoneum and pushed the latter upwards and inwards, by the aid of retractors we should obtain a good view of the bulging iliac fascia covering the abscess; this should be deliberately incised, and care taken that the wound in it is not made so long that the upper extremity is hidden under the subperitoneal fat that has been pushed up with the peritoneum. I mention this because it happens rather frequently, and is apt to lead to incomplete closure. The edges of the wound in the iliac fascia should be either held back with retractors or left untouched whilst the abscess and its contents are being dealt with; the seizing of them with artery forceps must be injurious in that it is apt to cause gangrene of the small parts of the wall they embrace. When the cleansing and drying of the cavity have been completed the opening in the iliac fascia should be most carefully and accurately sutured, a few sutures put in to approximate the split muscles, and the skin incision completely closed. Some years ago when the plan of complete closure of these abscesses was first practised, one of the causes of infection of the wound was, I am sure, neglect to thoroughly close up the opening in the iliac fascia; it was as often as not opened by a sort of puncture with a director and the opening enlarged with sinus forceps. The overlying muscles were sutured together but not this opening in the iliac fascia; this was left open and the deeper part of the wound was soon infected with tuberculous material leaking from the interior of the cavity. It is, I think, a mistake to open an ilio-psoas abscess in the loin even if it be presenting there; this used to be done frequently with the idea that better access to the interior of the cavity could be obtained; it is true that if one wished to drain these cavities an opening in this situation would be most useful as being in the most dependent position when the patient is lying on his back. But if the incisions are to be completely closed this argument fails; however carefully the deeper structures here are closed the earliest re-collection of fluid would lie immediately over the wound before that has had time to heal, and leak through it, and so infection of the wound would

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be almost certain to occur. The best place for the wound is in the highest part of the cavity when the patient is lying, so that it may have the best chance of becoming firmly united before fluid again comes in contact with it should re-collection occur.

Although it is now many years since this method of operating has been employed in cases of iliac abscess, it has been left to comparatively recent times to see its adoption in other parts of the spine, and I suppose almost the last to be dealt with in this way has been in the cervical region. Here drainage has been and, I believe, still is the general practice; but it is quite unnecessary, and abscesses in this situation can be treated in precisely the same way as those in the iliac fossa and with almost equally good results; a retro-pharyngeal abscess can be approached from the side of the neck behind the sternomastoid muscle, the contents cleared out, the cavity scraped and dried, and the wound completely closed, layer by layer, and union by first intention obtained. In some cases no re-collection of fluid occurs; should it do so in any quantity it can be dealt with from the opposite side of the neck, but should subsequent operations be necessary they must perforce be done through the site of one of the former ones. I am quite sure that any of you who may have had to deal with children under the two conditions will have fully appreciated the advantages to be obtained by this method without drainage.

It remains now to say a few words about those difficult and serious cases in which an abscess has formed in connection with disease of the lower cervical or upper dorsal vertebra, an important situation both because of the severity of the symptoms which may arise and of the difficulty of surgical treatment. On account of the latter if the symptoms be not really serious, I think it is best to leave them alone for a time. The patient may be kept with the trunk almost or entirely erect in a rigid jacket of some sort, with the hope—and sometimes the result—that the abscess will find its way down the front of the spine through the posterior mediastinum, and thence eventually into the psoas muscle, or to some situation where it can be more easily dealt with; and next to a caseation or drying up of the abscess, this is the best course we can hope for. If, on the other hand, the symptoms such as I have enumerated earlier are severe, and call for relief, something must be done, and to get at an abscess in this position there are three courses open. Those in connection with the lower cervical spine may sometimes be got at from the lower part of the neck by an incision behind the sterno-mastoid muscle—with perhaps division of a part of this—similar to that employed higher in the neck; but the operation is a difficult one, the structures among which the dissection is carried are important, and injury to them may be serious, whilst the room obtained when at last the abscess is opened is very limited. Another route by which the abscess may be reached is by performing laminectomy over the seat of the disease, and when the spinal cord has been exposed draw it gently aside and make an opening through the caseous bone in the body of the vertebra. This operation has little to recommend it except in cases where, in addition to the collection of fluid in front, there are signs of pressure on the spinal cord; in such rare cases the operation would be suitable, for it enables the two conditions to be dealt with at the same time. The third method available is by making a longitudinal incision in the back at a little distance from the mid-line, and removing the head and neck of one or more ribs, so approaching the abscess from the side of the body of the vertebra. And this on the whole seems to give the best results; the pleura has generally been lifted away from the side of the vertebra by the abscess, and so injury to it can be avoided; the intercostal nerve as it leaves the side of the vertebral column can probably be avoided, but even should it be injured or divided it would not be a matter of any vital importance. The only reason for not dealing with the interior of an abscess in this situation in the manner described before, and closing the wound, is the severity of the operation should it have to be repeated owing to a recurrence of the symptoms; but even this might be considered preferable to the risk of suppuration supervening in and about the diseased bone, which is more than likely to occur if prolonged drainage be resorted to.

I have sketched out what seems at the present time to be the most perfect way of dealing with one of the complications of spinal caries. Like those of any other surgical proceeding, the results are not invariably good, failures must occur sometimes—failure to keep the parts quite aseptic, failure to prevent the surface wound from becoming infected with tuberculosis; but I am glad to say success is the rule, failure the exception, and when by success we can keep these wounds aseptic and the patients comparatively free from pain, fever, or undue discomfort throughout what must necessarily be a long and somewhat tedious incapacity, it is a result which must be a source both of pleasure to the surgeon and of profit to the patient.

May 28th, 1906.

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WITH DR. SAMUEL WEST IN THE WARDS OF ST. BARTHOLOMEW'S HOSPITAL.

A Case of Granular Kidney and a Case of Diabetes with Threatened Coma.

When I brought my students to see this patient I asked them to try to tell me what was the matter with him by physical examination only, without asking any questions. As a matter of fact there was nothing to discover by physical examination except one thing, and that they came to last, and it might not have been discovered at all. I mean the condition of the vessels, which are much thickened. Although the tension is not very high to-day, it was at that time. The next thing which would in due course have been discovered would have been that there was a little albumen in his urine, and that, of course, would have suggested the diagnosis at once. There is no doubt that the patient has granular kidney, but he has not any albuminuric retinitis, notwithstanding the general arterial changes. And he does not look particularly ill. His arteries show that he has had mischief for a long time, and of course granular kidney is a disease which takes time to develop. You will find that he gives you no history of illness at all except occasional dyspepsia, which anybody might have, and which does not trouble him very much, and with this exception he says that he was perfectly well up to the very day that he was brought into the hospital. He came with a note from his doctor, who reported that when called to this patient, who was in business in the City, he found him vomiting and partly unconscious and complaining of very severe headache. The doctor prescribed for him and went away. But he had hardly reached home before he was summoned again, the message stating that the patient was in convulsions. The doctor diagnosed uræmia, bled him fairly freely, and sent him straight away to the hospital. When he arrived he was in a desperate condition, almost unconscious, and with occasional twitchings. He had vomited several times and was altogether in a critical state. He was half unconscious but could be roused. Having diagnosed the case as uræmia, we treated him actively, giving him a hot-air bath to make him sweat, with repeated doses of pilocarpin, about one twelfth grain each time, and two or three drops of croton oil on the tongue; and I left instructions that if he got worse and had actual fits he was to have inhalations of oxygen. As a result of the active treatment the condition quieted down. Next day he was practically convalescent and his symptoms had passed off. He now says he is quite well, and we shall soon let him go home. What I wanted to bring out about this case is the extreme rapidity with which symptoms of granular kidney may come on. I think I showed you last time a case of fits, which was not uræmic, but epileptic, also in a man with granular kidney. He had had a similar attack a month before, but previously to that date had never had any fits, and there was nothing to suggest a reason for the present attacks. Directly we examined him we found that he had thick arteries, albumen in his urine, and hypertrophied heart.

We do not know the cause of uræmia, and one of the problems which we have to solve is to explain why these attacks often come on without warning or apparent reason. We seem driven to suppose that the symptoms are due, not to any pathological lesion, but to some chemical substance which is developed suddenly in the body in some mysterious way. Of course that is not saying very much, but it is on chemico-physiological lines I think that we must seek for an explanation.

In association with this case I show you another, in which the disease is not granular kidney, but diabetes, and in which the symptoms also developed in an unexpectedly rapid way. It is a case of threatening diabetic coma. That patient also is now practically convalescent. When he came in he was vomiting, very dull and drowsy, and extremely feeble. We treated this case exactly on the same lines as the other. The only extra thing we did in this case was to give large doses of bicarbonate of soda. I do not know that that had any effect. I have never seen a case of well-developed diabetic coma saved, though cases which came under treatment before they were comatose I have seen recover. This case was treated on the theory that the condition was due to some chemical poison circulating in the blood which had developed in the same unexpected way as in the case of granular

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kidney. Coma has been treated by bleeding in diabetes just as in uræmia, but with little benefit. Pilocarpin was administered, the bowels were opened, and, as I have said in connection with the other case, he was to have had oxygen if he had become worse. It is interesting having these two cases together, and more interesting because both of them have been prevented from passing into the ultimate stage.

If there are chemical substances in the blood in diabetic coma, we might expect to find traces of them in the urine, and there are substances in the urine which have been supposed to stand in close association with diabetic coma. One of these is acetone, and another diacetic acid, but neither of them have really anything to do with diabetic coma. The diacetic acid (perchloride of iron) reaction is often present when the patient is not comatose, and it may be absent when he is comatose, or *vice versâ*; the same with acetone. They are both useful as indications, and show that the patient is less well than usual, and so might precede, but not of necessity, the onset of coma, but that is all. I have seen a diabetic patient who had quarrelsome relatives, and he always had an iron reaction in his urine after a visit from them. Acetone has been experimentally administered to animals in large quantities, and it has been given to man, but there were no symptoms of coma produced. If dogs are poisoned with acetone, they get a delirium like that produced by alcohol, but they do not become comatose.

Both patients were passing a large amount of urine, the diabetic 160 oz., and the uræmic about 120. It is difficult to make patients sweat when they have diabetes, but the pilocarpin did make him sweat, though not very profusely. I cannot understand how the statement has got into books that pilocarpin in kidney cases is a dangerous drug to use. It is a favourite remedy of mine, and I have never seen anything but good come of it. I give one twelfth or one sixth of a grain subcutaneously, and repeat it at intervals. Small doses at short intervals should be the rule for the administration of all strong drugs. When jaborandi first came into use it was generally the infusion that was given, which was of very uncertain strength, and contained many things which pilocarpin does not. I have seen patients who have taken infusion of jaborandi running from every aperture in the body—eyes, nose, mouth, bowels, skin, and bladder. You do not see those things after the administration of ordinary doses of pilocarpin, and in a large dose the bad effect produced is usually a faintness or collapse.

Morbus Cordis and Palpitation.

This little girl has morbus cordis, with a very dilated heart. Usually her heart is beating like a fire-engine. Full doses of Tinct. digitalis, 15 minims three times a day, and a belladonna plaster have quelled the heart's action. Although she has all this obvious throbbing she does not complain of palpitation. Patients who complain most of palpitation frequently have no organic disease of the heart. I think you may even go farther, and say when patients with morbus cordis do complain of violent palpitation there is very often a nervous factor in the case over and above the heart-lesion.

Case of Loculated Empyema.

This is a case of empyema which you saw when we last came round together. It proved to be loculated, so that the incision did not evacuate nearly all the pus that was known to be in the side. The day after operation a sudden fresh discharge took place, showing that another collection had made its way out by the incision. On admission, you will remember, the patient was spitting pus up as well, and the operation seemed to have little effect on this, and for a long time, although there was a free discharge from the wound, she still continued to spit up nearly half a pint in the day. Gradually that too has stopped, and the patient is now completely well. There must in this case have been at least three compartments in the pleura—the one which was opened, a second which subsequently discharged itself by the incision, and a third which evacuated itself through the lungs.

Alcohol and Tuberculosis.

Here is a middle-aged woman, who has syphilitic scars on the legs below the knee, a swollen abdomen, with dilated veins and a little fluid. There is some fluid in the left pleura, and possibly a little in the right pleura too. Her fingers distinctly show a fine tremor, and so does her tongue. She also has a raised temperature.

In her history there is not much, except the fact that she has been losing flesh and strength for the

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last six months. That is nearly all we can get from her. But I have to add to this that there is a difficulty in getting her to sleep. If you look at her, and take into consideration the fact that she has some fluid in her abdomen, with dilated abdominal veins, you will be led to diagnose alcoholic cirrhosis. The tremors of hands and tongue you can refer to alcohol. But what shall we say of her effusion into the left pleura, and possibly into her right pleura? In some cases of cirrhosis in the last stage general dropsy occurs. There is no general dropsy here, for the legs and feet are not œdematous. Dropsical fluid in the pleura ought not to cause a rise of temperature. So we are driven to the conclusion that the effusion is inflammatory, and, if inflammatory, very likely tubercular. The woman is evidently syphilitic, but syphilis has nothing, I think, to do with her present condition. So we are brought face to face with this question: Why has she become acutely ill just now? Is it not due to tubercular mischief? She denies being alcoholic, but her friends, on being pressed, confess it. So we may accept alcohol as an essential part of the case. I showed some of you a little while ago a case of a similar kind lying in No. 18 bed. That case was very much in the same general condition, and she was an alcoholic subject suffering from peripheral neuritis. She came in with great wasting, tremors, and a rise of temperature. We discussed the same question with regard to her, namely whether she had acute tuberculosis. We thought it was probable. However, we were wrong in that case, because at the autopsy she was found to have a recent, or subacute, consolidation, pneumonic in character, in her lung, which would account for her temperature. There were a few tubercular lesions in the lungs, but chronic, and apparently in a quiescent stage.

I think our present patient must have more or less acute tuberculosis.

There was at one time a belief that alcohol and tubercle were antagonistic, and it used to be stated positively that alcoholic persons did not become tuberculous. But that is not true, as we now know well enough. Reading such a *dictum* the other way round, it would follow that it would be good to allow tubercular cases large quantities of alcohol, a line of practice which was freely adopted at one time. However, the contrary is nearer the truth, and alcoholism seems in a way to predispose to tuberculosis—I presume by diminishing the resistance. Certain it is that many alcoholic patients die of tuberculosis. If you read the accounts of cases of alcoholic peripheral neuritis, you will find that the termination of many of them is by acute or subacute tuberculosis.

[The patient died shortly after this visit, and was found to have acute general tuberculosis, but the liver was fatty and not cirrhotic.]

Pernicious Anæmia treated with Adrenalin.

This is the woman with extreme anæmia whom you have seen before. She is getting better. She was in the hospital two years ago with the diagnosis of pernicious anæmia, and went out apparently well. When she came back, a few weeks ago, with a relapse the blood-cells numbered over 880,000. There being no tone in her vessels, I thought I would put her upon adrenalin. So she has been having 20 ♏ of adrenalin of a strength of 1 in 1000 lately three times a day. It is being gradually increased, and she has improved very much on it. Of course she has also been having raw meat juice, raw meat, and bone-marrow. But these things she had had before but without improvement until she took the adrenalin. The red blood cells are now half as numerous again as they were—that is to say, 1,200,000—and she is rapidly improving.

Some time ago I was asked what dose of adrenalin could be given with safety, and I was not prepared off-hand to say. One does not often see large doses of adrenalin given, and it produces a marked effect upon the vessels in small doses. I happen to have seen recently a patient who has been taking about two drachms or more of it in the course of twenty-four hours. That was a case of asthma, and it was because of the relief given in slight attacks that in a severe attack the doses were given repeatedly up to the amount specified.

Case of Hydrocephalus.

You have also previously seen this baby, which is the subject of hydrocephalus, and from which twelve ounces of fluid were removed; you can at once see that it is very much better—in fact, you would scarcely know the head again. It is three months old, and the hydrocephalus dated practically from birth. It had an enormously distended head, and

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we thought it was a suitable case for tapping. This was done, and we removed twelve ounces of fluid in about twenty minutes. All the bones of the skull fell together. We thought we had tapped one side and that the fluid from the other side had not drained. Accordingly the other side was tapped too, but we did not get much more fluid out. No harm resulted to the child from the procedure, but the skull slowly filled again. Then I raised the question whether we ought to perform some operation and establish drainage. The choroid plexuses secrete fluid which passes out of the ventricles, along the iter into the fourth ventricle, and so through the foramina of Munro and Magendie into the subarachnoid space; there is thus a regular circulation from the ventricles in the way I have described. If the circulation were stopped anywhere about the fourth ventricle the fluid would accumulate above, in the lateral ventricles, and hydrocephalus would result. If that be true, and a drain could be established between the lateral ventricles and the subarachnoid space, the circulation of the fluid might be established by a short cut, and the child might recover. We raised the question whether it could be done by surgical means. But the child was so ill that it was thought undesirable to interfere, and nothing was done. But from that time the child has been getting better, and the head is distinctly smaller. It is possible that as the result of the puncture a drain established itself. I do not know whether it would be possible to do an endocranial operation in this kind of case without laying the skull open, but in another case of this kind I should be inclined to try.

Post Mortems: (1) On the Case of Acute Caseating Pneumonia: (2) On the Case of Dysphagia.

I now want to say a few words about two post-mortem specimens. You will remember seeing a case of a man who had been ill only a short time. The upper part of the left chest was stony dull, he had a very high temperature, and was very ill. The case looked like pneumonia, only it had lasted too long, and there was such extraordinary dulness. After he had been in a few days we thought, knowing that the diagnosis of fluid was misleading, that it would be better to be certain there was no fluid there. Accordingly a puncture was made. A few ounces only of blood-stained fluid were obtained with the aspirator. On the next day pneumothorax was discovered, but I do not think the aspirator had anything to do with the production of it. However, the pneumothorax did not do any harm, for it produced no symptoms, and the air rapidly disappeared, although the patient was gradually sinking, and died three weeks afterwards. The diagnosis was interesting. At first pneumonia seemed probable because a little rusty sputum was brought up, in which nothing was found except diplococci. After this there was no sputum for some weeks. The diagnosis was made of acute caseating pneumonia, or acute pneumonic phthisis. It was not till some weeks later that a little sputum was obtained, and this, on examination, proved to contain numbers of tubercle bacilli, so that the diagnosis was confirmed. Here is the specimen; the whole left lung forms a solid mass like cheese, and it is one of the best examples of caseating pneumonia which I have ever seen. There are not many such specimens in the museums. The patient had been hoarse for two or three days before death, and the larynx showed small tubercular ulcers, while numbers of little follicular tubercles were scattered throughout the trachea and left bronchi.

The next specimen came from the man who had dysphagia. I will recall the main facts to your memory. He was a man æt. about 50 years, who said he had been losing flesh very much. He came in because of difficulty in swallowing, and his difficulty was a curious one, in that he could not swallow liquids but could swallow solids. If he tried to swallow a liquid it choked him, but if it were thickened by adding bread to it, or anything of that kind, it passed down all right. Examination of the chest revealed very little. In the right intrascapular region there was a small area of impaired percussion, where the vocal vibrations and vocal resonance were increased, and the air evidently had some difficulty in entering the right side. Therefore we concluded that he had some obstruction of his right bronchus. The cause of this might have been an aneurysm or a new growth. An aneurysm might have produced dysphagia, but it could not have produced this particular kind of dysphagia in which the patient could not swallow liquids but could swallow solids. Therefore we were bound to conclude that he had something which led to a perforation between the œsophagus and the trachea,

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and that the perforation could not be a large one, and could only have been produced by a new growth and not by aneurysm. Here is what we found. An extensive malignant ulceration of the œsophagus involving the root of the right lung, with two small openings into the trachea. You will remember that the man was bringing up about a quart of muco-purulent secretion daily, which must evidently have come from his trachea. This was explained post mortem by the infiltration of the trachea by the new growth and by the perforations I have mentioned.

May 28th, 1906.

THE TREATMENT OF PILES, PROLAPSE, AND PROCIDENTIA RECTI.

By DOUGLAS DREW, B.S., F.R.C.S.,
Surgeon to the North-Eastern Hospital for Children and to the Hospital for Women.

THE operative treatment of piles is a subject which, like a great many other simple surgical questions, has received a large amount of attention at the hands of many writers. Much controversy has centred around the best method of dealing with this distressing malady, and endless varieties of clamps, forceps, etc., have been designed as aids in the performance of some new modification of the operation.

The various methods in vogue at the present time may be summarised in three main groups, each of which has its ardent advocates:

(1) Clamp and cautery.
(2) Ligature.
(3) Excision.

The first of these methods was introduced by Henry Smith, who invented a clamp for the purpose, which is still used by those who employ this method of operating. In performing the operation, the pile is seized in the clamp, which crushes it, and after cutting a portion of the pile away, the stump is cauterised. It is claimed for this method (1) that hæmorrhage is doubly guarded against, first by the crushing and then by the cauterising; (2) that septic absorption is prevented by the eschar and that the edges of the wound are in apposition and glued together.

In the second method, by ligature, a notch is cut in the mucous membrane at the base of the pile, and it is separated from the submucous tissue. A ligature is then applied to its base, and the distal portion is cut away, or left to slough off with the ligature. This method was strongly advocated by the late Mr. Allingham, and it is frequently described as "Allingham's method," although it was originally introduced by Salmon. It is perhaps the operation that is most frequently practised at the present time, owing, I think, in large measure, to the able advocacy of the late Mr. Allingham, and to the fact that the operation is extremely simple to perform.

Whichever of these methods is adopted the result is much the same—*i. e.* the wound heals by granulation, and even allowing that the sphincter by its contraction tends to approximate the edges of the wound, yet the process of healing cannot be so rapid as when the edges of the mucous membrane are accurately adjusted by suture after excision of the pile. It is somewhat strange that, in this connection, the important surgical principle of endeavouring to obtain union of a wound by first intention has been so largely ignored. Yet there is no more reason that a wound in the rectum should not heal by first intention than one in the mouth if it is treated in such a manner as to render this possible.

Bearing this principle in mind, I have for some years past treated all cases of piles by excision and suture, varying the procedure according to the requirements of the case.

In a simple case, where there are two or three globular piles, after thoroughly stretching the sphincter and cleansing the rectum, the pile is seized in forceps, and the mucous membrane is divided vertically upwards; or if the pile is continuous with one situated externally, the skin at the base of the latter is divided as described, and the incision is extended upwards by successive snips with the scissors, the edges of the wound being sutured with fine catgut, which will absorb in ten days. Before completely severing the pile, a suture is passed beneath the undivided part, so that, on tightening this suture, the vessel running into the pile is secured. On completion, the vertical row of sutures may be felt within the bowel. The operation is performed without any clamp, and there is practically no bleeding.

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Where the piles are numerous, and there is much prolapse of the mucous membrane, I invariably perform Whitehead's operation, which appears to me to be the only method which is suitable for these cases by which a permanent cure can be expected.

Whitehead originally introduced his operation of complete excision of the pile-bearing area of mucous membrane in 1882, and his method, or some modification of it, has been largely adopted by many operators. This method, however, was very strongly criticised by Allingham and others, and many objections urged against it. The chief, and the one which, in my opinion, has the slenderest foundation in fact, is that stricture of the anus is very prone to develop, owing to the stitches giving way, and retraction of the mucous membrane occurring. While admitting that such a result is possible if the mucous membrane is removed too freely, I have never seen anything of the kind occur, although I have performed the operation a large number of times. The only cases of post-operative stricture of the anus (two in number) that have come under my observation have resulted from the application of ligature, and one of them was of a very severe and intractable nature.

Another objection often urged against Whitehead's operation is that it is a much more serious procedure, and that the loss of blood is excessive. While admitting that the operation on the average takes about half an hour, it has not been my experience that the loss of blood has been excessive or in any way injurious. The advantages of excision more than compensate for the time and trouble expended. The wound is usually completely healed in from ten to fourteen days. The whole of the pile-bearing area is removed, so there is no risk of return of symptoms.

On careful digital examination the scar can with difficulty be felt situated about a quarter of an inch inside the anus.

After the application of the ligature the process of healing takes longer, owing to the fact that a very considerable portion of the circumference of the bowel is denuded of its mucous membrane and left to granulate, and as the process of granulation proceeds contraction takes place, which, although only exceptionally developing into a stricture, yet necessitates the occasional introduction of a bougie or the finger, which causes the patient considerable distress. Allingham recommends the daily introduction of the finger after the first week in order to avoid contraction. It is not at all uncommon to find that the healing is not complete at the end of three weeks, while in the end there is always a risk that some of the smaller piles, which may not have been removed, may gradually develop and cause a return of the symptoms. I have had on several occasions to perform secondary operations after ligature of piles.

Although Whitehead's operation is not difficult to perform, yet it is by no means such an easy operation as ligature, and it is, perhaps, owing to this fact that its degree of popularity has been limited.

In performing the operation certain points require careful attention.

The sphincters must be thoroughly stretched and the rectum cleansed.

The incision should be made accurately at the muco-cutaneous margin, and must not be allowed to extend outwards so as to include any tags of skin around the anus, otherwise it will be found that after the operation is completed the mucous membrane will be drawn outside the anus. If any of these tags of skin appear to require removal, it may be done by snipping them off with scissors by an incision radiating from the anus, and the edges are united by catgut sutures, but for the most part they do not require to be cut away.

The mucous membrane carrying the piles is dissected up the required distance. The ease with which this may be accomplished will depend upon whether the patient has had any previous attacks of inflammation in the part. If so, it will be found that there is a varying amount of fibrous tissue which will require to be divided before the mucous membrane can be raised. But in the absence of old inflammation the mucous membrane can be stripped up with dissecting forceps with the greatest ease. In doing so the sphincter will be exposed. It is important to clearly define this structure so that there may be no risk of removing any of its fibres.

The mucous membrane is removed by successive snips of the scissors and the edge united to the skin, care being taken to adjust the corresponding part of the skin and mucous membrane so that no redundance of either may be found when the last portion comes to be sutured.

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The term "prolapse" is used in a very loose manner to describe conditions which are essentially different—on the one hand, to describe a protrusion of the mucous membrane of the bowel, involving a part or the whole of the circumference, a condition which in the adult is usually associated with piles; while, on the other hand, the term is used to designate a complete falling or protrusion of the whole rectal wall, a condition not uncommonly met with in children, but which is decidedly rare in adults, except in advanced years. To this condition the term "procidentia" has been applied to distinguish it from prolapse. Other writers differentiate between the two conditions by describing the protrusion of the mucous membrane as "prolapsus ani," while to the second and more serious condition the term "prolapsus recti" is given.

The important point is to be able to distinguish between the two conditions clinically, and to recognise that, if any operation is performed, in the one case only the mucous membrane is protruded, while in the other the mass is composed of the whole of the rectal wall, and if removal is attempted the abdominal cavity will be opened.

Many writers regard procidentia recti as an aggravated degree of prolapse of the mucous membrane. They consider that the dragging on the rectal wall by the prolapsed mucous membrane will gradually produce a protrusion of the whole wall.

When prolapse arises from a polypus attached to the rectal wall above the sphincters, it is possible that the rectal wall may be dragged down, but that prolapse of the mucous membrane associated with piles ever produces such a condition I think is extremely doubtful.

In adults, prolapse of the mucous membrane is extremely common, and if one excludes cases which arise from inflammation, and are of a temporary nature, and those cases in women arising from a torn perineum, the condition is almost invariably associated with hæmorrhoids.

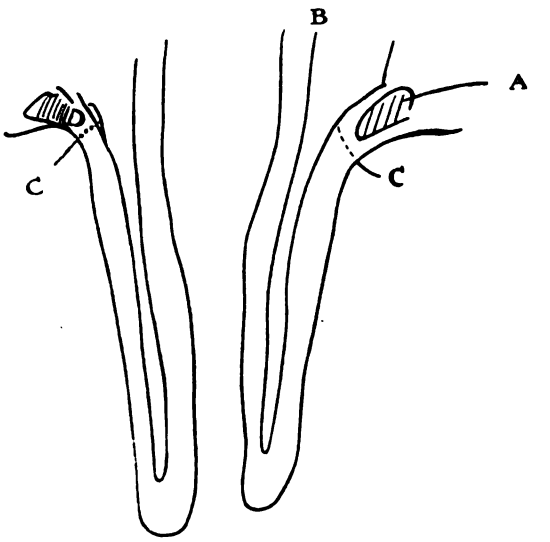
Procidentia is *ab initio* a falling of the whole rectal wall, and is brought about by any condition which tends to increase the intra-abdominal tension, and is oftentimes associated with a weakness of the structures composing the pelvic floor or loss of support below from a ruptured perineum.

The treatment of prolapse of the mucous membrane has already been described.

The treatment of procidentia is a much more serious and difficult matter, except in children, where the condition is met with in weakly and rickety children, and is often determined by an attack of diarrhœa.

Occasionally one meets with a case in a child in which all the ordinary methods fail to effect a cure. Such cases may be treated by linear cauterisation, after which the mass is reduced within the anus. I have, however, found it a better plan to remove one or more lenticular-shaped pieces of mucous

FIG. 1.



Procidentia recti. A. Sphincter. B. Peritoneum. C. Line of incision.

membrane from the prolapsed part and to suture the edges.

In the adult this measure is as futile in effecting a cure as cauterisation, and removal of the whole mucous membrane from the protruded portion has been practised with little better success.

Another method of treatment is to endeavour to prevent the bowel from coming down by narrowing the anus by excising elliptical pieces of skin and mucous membrane. The results attained by this measure have proved to be only temporary.

By far the most promising method is to treat the mass by excision. This I have done in two cases in women, æt. respectively 39 and 41. The condition had existed since childhood, and in both cases a previous operation had been performed without success. The mass in each case protruded several inches from the anus. The mucous

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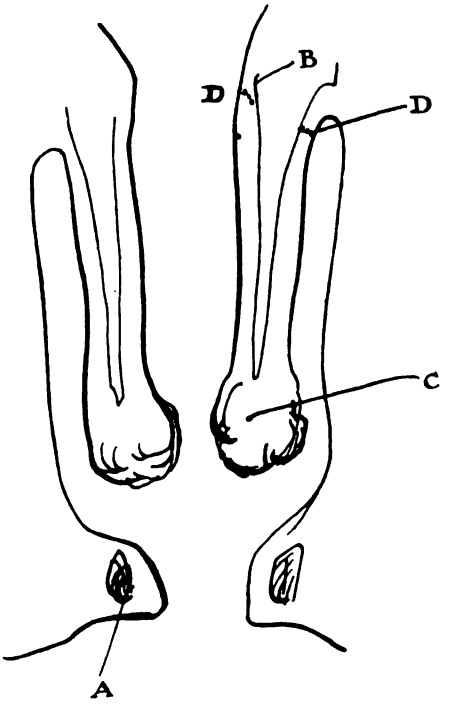
surface was in places ulcerated, and reduction was painful and difficult. Owing to the length of time that the condition had existed, the sphincters were completely stretched by the large protruding mass, and it was anticipated that after removal the sphincter power would remain impaired, and that incontinence of fæces might persist. This assumption, however, I am glad to say has proved incorrect, as both patients have considerable contractile power in the sphincter, and although this weakness is present in one case when the bowels are relaxed, yet under ordinary circumstances there is no inconvenience. This case derived considerable benefit from faradism applied locally after the wound was healed.

FIG. 2.



Intussusception of rectum. A. Sphincter. B. Peritoneum. C. Growth. D. Line of section.

The method of operating was as follows: After thoroughly cleansing the mass, both within and without, an incision was made, just as in Whitehead's operation, at the muco-cutaneous junction. The sphincter was defined and separated, and the dissection deepened in front until the peritoneum was reached and opened. On each side the edges of the levator ani were defined.

The opening in the peritoneum was temporarily plugged with gauze, while the bowel was freed behind, and the vessels in the meso-rectum were secured as they were divided, after which all the loose bowel was drawn down. The peritoneum was next united to the bowel by a continuous stitch, about two inches above the point at which the bowel was to be divided. The bowel was then cut way, and the divided edge accurately sutured to the edge of the skin.

In one case nine inches were removed, while in the second fifteen inches were removed. In both the convalescence was satisfactory and the results excellent. I have recently seen both patients (ten and twelve months have elapsed since the operation). There is no tendency to recurrence of the condition, and the scar is so slight that it would be quite easy to overlook it altogether in examining the rectum.

Another and rare variety of prolapse of the rectum is occasionally met with in which the lower segment of the bowel retains its normal position, instead of being involved in the prolapse as in the cases described above. In this variety the upper part of the rectum or sigmoid descends into the lower part and may actually protrude from the anus. Such cases are often described as intussusception of the sigmoid, and are usually occasioned by stenosis of the bowel from growth or some other cause of narrowing, such as scarring from ulceration.

I have operated upon one case of this nature in which a ring of carcinomatous growth formed the apex of an intussusception, which was situated just above the sphincter. The method of operating consisted in excising the intussusception with the growth by dividing the returning layer at its junction with the rectal wall, and suturing the entering layer to the cut edge of the rectal wall step by step as it was divided. In this case the union was satisfactory. The patient, however, died from internal recurrence of the growth within eighteen months of the operation (Fig. 2).

The specimen, showing the growth and the portion of the rectum removed with it, was presented to the Royal College of Surgeons' Museum.

May 28th, 1906.

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THE OPERATIVE TREATMENT OF CAVITIES IN THE LUNG.*

By JAMES BERRY, B.S., F.R.C.S.,

Surgeon to the Mount Vernon Hospital for Consumption and other Diseases of the Chest; Senior Surgeon to the Royal Free Hospital.

GENTLEMEN,—On a previous occasion when I had the honour of lecturing to you I chose for my subject a disease of the chest in which surgery affords practically the only means of treatment. It is the commonest of all diseases of the chest which the surgeon is called upon to treat. It is one of which all of us must have had considerable practical experience. I refer, of course, to empyema. To-day I take for my subject cavities within the lung itself, diseased conditions which from the surgical point of view are distinctly rare. Either the condition is a common one, which but rarely calls for operative treatment, or the condition urgently demands operative interference, but is of rare occurrence.

Of the first a tuberculous cavity is an obvious example; of the second an abscess following pneumonia, or, still more important, the abscess or gangrenous condition of the lung following impaction of a foreign body in the bronchus.

It is, then, with cavities within the lung and their appropriate surgical treatment that I propose to deal to-day. The individual experience of any one surgeon in this branch of surgery can seldom be very large, since opportunities for operating upon the lung for disease are but seldom afforded to an individual surgeon. My own individual experience of operations upon the lung itself (for disease) has been small and limited, occasional operations for abscess and gangrenous conditions following pneumonia or due to the impaction of a foreign body in the bronchus. I

* One of the Mount Vernon Hospital Course of Post-Graduate Lectures.

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shall make no excuse, therefore, for drawing largely upon the works and experience of others who have written upon this very interesting and difficult branch of surgery.

My object to-day is mainly to give a general account of what has been done and what may be done in this field. Numerous monographs, numerous isolated papers upon pulmonary surgery, have been published. Perhaps the most valuable among recent sources of information are the consecutive series of cases taken from one or more clinics, such as that published in 1902, by Garré and Sultan, from the Kœnisberg and Rostock clinics (20 cases), that of Borchert from the Urbankrankenhaus of Berlin (21 cases, 1890-1900; or, including cases of secondary empyema, 29 cases). Among recent monographs dealing largely with series of collected cases one of the best is that of Tuffier, 'Chirurgie du Poumon,' 1897, which contains some hundreds of cases of lung surgery, mostly given in abstract only.

I must also acknowledge my indebtedness to the admirable standard works of Fowler and Godlee on 'Diseases of the Lungs,' 1898, and Samuel West 'Diseases of the Organs of Respiration,' 1902, and various other authors.*

The various cavities within the lung which are more or less directly amenable to surgical interference may be arranged as follows:

(1) Abscesses and gangrenous cavities following upon pneumonia.

(2) Bronchiectatic cavities.

(3) Tuberculous cavities.

(4) Chronic non-tuberculous cavities caused by abscesses bursting into the lung from outside (*e.g.* abscess of the liver).

(5) Cavities caused by foreign bodies.

(6) Hydatid cysts.

A further classification of some importance from the surgical point of view is into acute and chronic cavities. The situation of the cavity is also of much importance. Cavities at the base of the lung and cavities at the surface are much more accessible, and therefore more easily operated upon, than those at the apex and those which are deeply seated towards the root of the lung.

"Surgical interference with the lung," says Tuffier, "demands exact diagnosis, bearing upon the existence, the nature, the seat, the shape, and number of the lesions in the affected lung, upon the state of the pleura, of the opposite lung, and of the general visceral condition of the patient."

Unfortunately, exact information upon all these points is not always obtainable. Even the most skilful physician is not always able to indicate to the surgeon the exact size and situation of the cavity which he asks him to open. With regard to the existence of pleural adhesions, a matter of the greatest importance to the surgeon who is about to operate upon the lung, the physician cannot always tell him with certainty that they are present.

The surgical methods of treating pulmonary cavities may be divided into three groups.

(I) *Intra-pulmonary injections* by means of a hollow needle introduced through the chest-wall. By this means various antiseptic substances have been introduced directly into cavities, usually tuberculous vomicæ. The experience of those who have employed this method (I myself have no personal experience of it) seems to be that they are of but little use and not free from danger. Speaking of intra-pulmonary injections for tuberculous cavities, Dr. Samuel West says that this method of treatment "has done no good, but, on the contrary, in some instances, by exciting fresh inflammation, has done positive harm.*

"Little is to be hoped from intra-pulmonary injections" is the verdict of Mr. Godlee† on the same subject.

(II) *Excision* of diseased portions of lungs has been attempted on many occasions for tubercle, although more often for the early stage of tubercle than when the stage of cavitation has been reached. Usually the apex of one lung has been removed through an opening produced by removing the second rib. Even the whole lung has, on one occasion, been removed without causing the death of the patient. It need scarcely be said that this practice is not to be recommended, on the ground of the great danger of the proceeding in those who are subjects of tubercle, and the great improbability of doing any good whatever thereby.

Sarfert, of Berlin, after much study and experiment upon phthisical cavities in the dead body,

* A very useful list of references to no less than seventy papers on subjects connected with the surgery of the lungs is given by Dr. J. Schutz in the 'Centralblatt f. d. Grenzgebiete d. Med. u. Chir.,' January 1st, 1901.

* 'Diseases of Lungs,' p. 428.

† 'Diseases of Respiratory Organs,' vol. ii, p. 544.

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has devised and described an extra-pleural method of shelling out the apex of the lung. He gives several photographic illustrations of the operation, and in a supplement to his paper describes a clinical case in which he performed it on the living. His monograph* on the operative treatment of consumption contains a good deal of information interesting to those who desire to perform operations upon apical phthisical cavities. Briefly, the proceeding is as follows: The second rib is removed, the dome of the parietal pleura is then separated from the thoracic wall, and the apex of the lung with both layers of pleura intact is then drawn forwards into the wound and fixed there. The apex of the lung can then be carefully examined by palpation. The existence and exact situation of a cavity can be definitely localised, and suitable surgical treatment to the lung itself can thus be carried out (incision, drainage, removal, etc.).

(III) I come next to the third method of treating pulmonary cavities—namely by *incision and drainage*. This is the only surgical method of any real practical importance.

In the vast majority of cases when a surgeon operates upon a diseased lung he does so in order to drain a cavity. That cavity may occasionally be a non-suppurating one, as in the case of a hydatid cyst, but it is almost always a suppurating one. The cavity may be a closed one, as in the case of an unruptured hydatid or abscess; but more often it is one that communicates more or less imperfectly with the interior of the bronchi.

Let us now consider a few points in connection with the above-mentioned six classes of cases in which surgical interference by incision and drainage may be demanded.

(1) In cases of abscess and gangrene following pneumonia the cavity should, as a rule, be opened if the diagnosis can be made. "The recognition of a local lung destruction, gangrenous or otherwise," says Babcock,† "depends not only on physical examination of the chest. Indeed, in some cases this may furnish less information than the history and symptoms."

These patients are usually extremely ill, and the circumstances under which the operations have to be performed, especially in the case of gangrene, are extremely unfavourable.

Of abscesses in general treated by operation, Garré and Sultan have collected 91 cases from literature, and adding 5 cases of their own (of which 4 recovered and 1 died), they present us with a series of 96 cases, of which 77 were cured (80 per cent.), and 19 died (20 per cent.).

In gangrene the mortality is naturally much higher. Of 122 cases collected by the above-mentioned authors, including 6 cases of their own, 80 were cured and 42 died.

The following is a typical case of a large abscess of the lung following pneumonia, and successfully treated by operation.*

Frederick J—, æt. 34 years. In the year 1892 he was suddenly attacked with cough and pain in the right side, fever, and expectoration. There was no hæmoptysis. After an illness lasting four weeks (pneumonia) he recovered. Two years later he had a similar illness, lasting twelve weeks, from which he also recovered. Two years later he was again seized with similar symptoms, together with frequent hæmoptysis. This time, however, he did not make a good recovery. Pain, cough, expectoration, and fever continued. The patient lost flesh and had night-sweats. For some months the expectoration was profuse, purulent, and foul-smelling. On admission to hospital he was in a bad state of nutrition. Temperature 100·4° F. The percussion note over the right apex was diminished. From the fourth intercostal space downwards there was marked dulness. In the axillary line the dulness began at the level of the sixth rib; at the back it began at the seventh rib. After violent coughing the percussion note became clearer and the area of dulness smaller. Ascultation over the right apex showed vesicular breathing. Over the dull area there was bronchial breathing, with amphoric sounds, and near it coarse *râles* could be heard. All these physical signs were most clearly marked two fingers' breadths below the angle of the scapula. The bronchial breathing was more

* 'Die Operative Behandlung der Lungenschwindsucht,' Berlin, 1901.

† "The Diagnosis and Differential Diagnosis of Pulmonary Abscess and Gangrene, with a View to Surgical Treatment," by Rob. H. Babcock in 'Journ. of Amer. Med. Assoc.,' June 18th, 1898, an excellent article on the subject.

* Garré and Sultan, 'Kritischer Bericht ueber 20 Lungenoperationen Beitr. z. Klin. Chir.,' 1902, vol. xxxii, p. 507.

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marked after prolonged coughing. The sputum was abundant, thin, purulent, greenish, and stinking. There were no elastic fibres in it but many bacilli "resembling those of tubercle." Puncture two fingers' breadths below the angle of the scapula gave exit to pus with characters above mentioned.

On February 15th, 1898, five days after the puncture, Prof. Garré operated. He resected about two inches of the posterior parts of the eighth and ninth ribs. The pleura was found to be much thickened and its cavity obliterated. After demonstration of the presence of pus by a second puncture, an incision was made with a Paquelin's cautery and knife. At a depth of nearly an inch (2 cm.) an abscess cavity was opened and 500–600 cubic centimetres (about a pint) of stinking pus was let out. The abscess cavity was twice as large as a man's fist, and had a smooth lining. Opening into it were four large bronchial tubes, and from these pus was observed to flow. The edges of the opening in the lung were drawn down and sewn to the thoracic wall, and the cavity packed with sterile gauze.

The operation was begun under chloroform, but its administration had to be discontinued towards the end as the contents of the abscess came partly through the nose and mouth. The patient was somewhat collapsed, but quickly recovered.

The cough, expectoration, and secretion from the wound soon began to diminish, and the latter quickly became less foul.

The packing was removed on the third day and replaced. On one occasion, a fortnight after the operation, the temperature rose to 104·3° F., probably from the packing being too tight. The wound was kept filled with gauze, and soon contracted up.

Six weeks after the operation the patient was able to go home. The wound was then small and superficial. Examination of the granulations and secretion from the wound was negative as regards tubercle bacilli.

There were no abnormal breath sounds except a few râles in the immediate neighbourhood of the wound.

The report of the patient's condition at the end of October, 1901, was very satisfactory. He had completely recovered, he felt very well, and was able to do all his ordinary work.

The chief points to notice about the diagnosis of this case are the localised area of dulness and bronchial breathing; the fact that after violent coughing the area of dulness became smaller, and the breath-sounds more audible, indicating the partial evacuation of a fluid-containing cavity. The continued cough, pain, fever, and profuse blood-stained, foul-smelling, expectoration all pointed to abscess. The smooth walls of the cavity and the absence of tubercle bacilli from the granulations lining it were against the tuberculous nature of the lesion in spite of the doubtful discovery of tubercle bacilli on an earlier occasion.

From the operative point of view a very important point is the fixation of the lung to the chest wall by means of sutures. If this be not done in such a case, it is not unlikely that the lung will recede from the chest wall and a consecutive pneumothorax, with all its grave risks, be produced.

In one case of my own, in which I had to open a very large gangrenous cavity in the lung of an elderly man, I did not take this course and had cause to regret that I did not. The patient was extremely ill and the adhesions between lung and thoracic wall seemed so firm that I was tempted to trust to them and not prolong the operation by the insertion of sutures. Partial retraction of the lung took place, permitting escape of stinking pus and air into the pleura, and the patient died of septic absorption about a week after the operation. I think it possible that this patient might have recovered had I adopted the precaution above mentioned.

Prof. Garré, in his comments upon his own case, remarks that the resection of ribs might with advantage have been more extensive, and that had the opening into the abscess been somewhat larger, healing of the cavity would probably have taken place more quickly.

(2) *Bronchiectasis.*—The results of operations for bronchiectatic cavities can scarcely be said to be encouraging.

The patients are generally in extremely bad health by the time that a bronchiectatic cavity is large enough to be diagnosed and its position localised. Such cavities rarely attain a large size and are usually multiple. The conditions are not therefore usually suitable for satisfactory drainage. On the other hand, the wretched condition of some of these patients, much distressed with the frequent expectoration of quantities of stinking muco-pus, sometimes tempts the surgeon to endeavour to

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alleviate the condition by operative procedures. Of the five cases recorded by Borchert four died, and of a similar number in Garré and Sultan's series three died shortly after the operation and a fourth a few months after a second operation for the same condition.

The statistics of cases collected by these authors from literature (always an untrustworthy source of information as regards mortality) gave fifty-seven operations with twenty-one deaths.

(3) *Tuberculous cavities.*—Here again the results of operations must almost necessarily be unfavourable on account of the very unfavourable general condition of patients with phthisical cavities.

Occasionally a simple large cavity, especially at the base of the lung, may be drained with advantage, if the rest of the lung can be shown to be healthy or nearly so, if the opposite lung is sound, if the general condition permits of an operation, and if the symptoms and physical signs indicate that drainage of the cavity is likely to be of benefit by facilitating the escape of septic discharge.

It is possible that apical cavities might with advantage be opened more often than they are. Mr. Godlee, writing in 1898, expressed guardedly the opinion that "in a certain number of cases with single apex cavities and with troublesome cough not only might the symptoms be relieved, but the danger of transferring the mischief into the other lung might be to some extent lessened."

I will here give an abstract of a published case of apical cavity operated on by Sarfert in the manner already described (Sarfert's case, phthisis, apical cavity, operation). *

Married woman, æt. 46 years. Ill one year with cough, expectoration, hæmoptysis (more than once), and pain in the chest.

July, 1899.—Dulness above and below right clavicle and down to third rib. Auscultation over the same region showed well-marked amphoric breathing, and other signs of a cavity. The rest of the right lung and the whole of the left lung were practically sound. Expectoration contained elastic fibres, tubercle bacilli, and other bacteria and cocci. Afternoon temperature, 101·5° F. Night sweats. No albuminuria.

September, 1899.—More hæmoptysis (tumblerful). Temperature rather higher.

October, 1899.—Several rigors, expectoration much more profuse.

Diagnosis.—Single large phthisical cavity at right apex, and probably pleural adhesions.

December 16th, 1899.—Operation. Removal of most of second rib. Pleura adherent. Apex of right lung and dome of pleura separated without trouble. Cavity packed with iodoform gauze. Operation well borne. No asphyxial symptoms.

December 20th.—All gauze removed. Palpation of the exposed apex (covered with parietal pleura) revealed a soft area, the site of the underlying cavity. This was punctured and then incised. Very trivial bleeding occurred. The cavity in the lung was found to be as large as a hen's egg. The wound was packed at first with iodoform gauze, and then with plain sterile gauze. The patient made a good recovery from the operation, and soon noticed that cough and expectoration were both much less.

January 6th, 1900.—Patient allowed to become an out-patient. Wound dressed daily. Temperature (taken four times a day) henceforth varied between 98·8° and 100·2°. The wound cavity gradually filled up with granulations.

By March 15th, 1900, the operation wound had diminished to a superficial area three fingers broad, in the middle of which was a sinus a little more than an inch long, with a blind extremity.

Sarfert, in commenting upon this case, alludes to the ease with which the operation was carried out and the little hæmorrhage that occurred during its performance.

I have given the case at some length because operations upon phthisical lungs are not common. If an operation upon an apical tuberculous cavity is ever justifiable, it would seem to me to be in just such a case as this, where pain, hæmoptysis, and signs of septic absorption are all well marked, where the patient's general condition is fairly good but is steadily deteriorating, and where the signs of disease are well marked at one apex but absent from other parts of the lungs.

That some relief at least was given to the patient seems clear from the account of the diminution in the amount of expectoration and cough and the marked lowering of the temperature. It is a pity that the report of the case ends three months after the operation, and that we are not informed of the subsequent history of the patient.

* 'Die Operative Behandlung der Lungenschwindsucht, Appendix,' H. Sarfert, Berlin, 1901.

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(4) An interesting case of abscess of the lung, which would seem to fall into the class of non-tuberculous cavities bursting into the lung from the liver, is recorded by Dr. Watkins-Pitchford.* The exact origin of the abscess was not, however, quite clear. A soldier, æt. 29 years, who on three separate occasions between the years 1885 and 1900 had sustained severe injuries to the right side of the chest from falls from a horse, began to expectorate blood-stained pus two days after the last fall. Cough and expectoration continued for three months. He was then admitted to the General Hospital at Pretoria. At that time "the expectoration had for many weeks averaged about three pints in each twenty-four hours," and cough was almost "incessant." "The right side of the thorax was tender to percussion, the greatest tenderness being over the fifth rib, outside the nipple line. Surrounding the right nipple was an area about the size of the palm of the hand in which the percussion note was very dull, breath sounds absent, voice sounds much diminished, and vocal fremitus abolished. Below, this area became continuous with that of the liver dulness. Over the base of the lung behind and at the side percussion resonance was impaired and breath sounds extremely faint. Percussion resonance over the upper part of the lung was skodaic. No râles could be detected in the right lung, and the physical signs of the left were normal. The heart gave signs of being displaced three quarters of an inch to the left; its sounds were natural. No enlargement or tenderness of either liver or spleen could be detected. The expectoration, which was of a bright pink colour, when examined microscopically, proved to consist of much ill-defined granular *débris*, a few pus-cells, and many red blood corpuscles. The tubercle bacillus could not be discovered, although searched for on two or three occasions."

The diagnosis made was, "A collection of fluid near the base of the right lung, either localised in the pleural cavity or contained within the substance of the lower lobe."

An operation was performed by Major Porter, R.A.M.C. After exploration with a needle had shown the presence of pus at a depth of three inches from the surface, a portion of a rib was excised, the lung exposed, and about four and a half ounces of thick, grumous, non-offensive pus was evacuated from a cavity "large enough to hold the closed fist and surrounded on all sides by lung substance."

The patient made an excellent recovery. The chief point of interest after the operation was the discovery, a few days later, of bile both in the expectoration and in the discharge from the wound. Dr. Watkins-Pitchford discusses fully the interesting question of the mode of origin of the abscess, whether from the original injury to the lung or from the liver. He remarks that the patient had "never suffered from pain in the region of the liver, jaundice, or dysentery," but says that the presence of bile in the discharge from the abscess after the operation is difficult to explain except by the theory of the abscess having being primarily in the liver.

The subject of abscess of the lung caused by foreign bodies impacted in the air-passages, as well as that of hydatids in the lung, I propose to reserve for a subsequent lecture.

June 4th, 1906.

* 'Brit. Med. Journal,' April 20th, 1901, where the case is reported in full.

CLINICAL LECTURE

ON

THE ETIOLOGY AND PROGNOSIS OF NEURASTHENIA,

ESPECIALLY IN ITS RELATION TO GASTRO-INTESTINAL CONDITIONS, BASED ON AN ANALYSIS OF 103 PRIVATE CASES.

By THOMAS D. SAVILL, M.D.Lond.,
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GENTLEMEN,—The examination of the stools is a difficult and unpleasant task. Nevertheless, I believe that if it could be done more frequently and more thoroughly considerable light would be thrown on the pathology of alimentation, on the various forms of toxæmia, and, in particular, on the pathology of neurasthenia.

I have on previous occasions* dwelt on the

* 'Lectures on Neurasthenia,' 1st edition, 1898; 2nd edition, 1902; 3rd edition, 1906. London: H. J. Glaisher. See also the CLINICAL JOURNAL, February 24th, 1897.

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important relation which various gastric disorders bear to neurasthenia among hospital out-patients, and for some years I have been inclined to believe that various intestinal conditions allied to mucous colitis play an equally important *rôle* in the etiology of neurasthenia, but the opportunity of testing this belief has been wanting in hospital practice.

To ascertain the truth in this matter it would be necessary to inquire, first, what proportion of cases of neurasthenia have been preceded by symptoms of intestinal derangement, and, secondly, whether adequate treatment adopted for the relief of the latter also cured or relieved the neurasthenic symptoms.

Now, a hospital out-patient clinique such as I have availed myself of on previous occasions does not afford very good material for arriving at a conclusion on these points. The symptoms of intestinal derangement are of a kind which escape the notice of the ordinary hospital patient, who does not, as a rule, even if he is sufficiently intelligent, observe the character of his motions; the time or opportunity in out-patient practice is not sufficient for a physician to examine these cases thoroughly for obscure intestinal troubles; and finally, the treatment for mucous colitis and other intestinal conditions cannot be carried out with sufficient diligence or care to afford reliable data. With private patients, however, all the conditions are more favourable, and I propose now to avail myself of this field to investigate the two points just mentioned.

During the three and a half years February 27th, 1902, to August 18th, 1905, 103 cases of neurasthenia came under my notice in private practice. There were 55 male cases and the average age of these was 40½ years, the youngest being 14 years and the oldest 62 years of age. There were 48 female cases, and the average age of these was 38 years, the youngest being 16 years and the oldest 71 years. Taking male and female cases together, there were:

3	in	the	decade	10	to 19
20	,,	,,		20	,, 29
33	,,	,,		30	,, 39
28	,,	,,		40	,, 49
16	,,	,,		50	,, 59
2	,,	,,		60	,, 69
1	,,	,,		70	,, 79
Total 103					

Among these cases, therefore, as in my hospital series (*loc. cit.*, 2nd edit., p. 67), there was a marked tendency for neurasthenia to affect the early and middle decades of life (81 cases being in the three decades 20 to 49), and a predisposition in the male sex, 55 as compared with 48.

These 103 cases of neurasthenia which it is now proposed to analyse from an etiological point of view were all of those which I met with in private practice between the dates mentioned; they are in series and unselected, and none have been excluded. They all came under notice for the symptoms of neurasthenia.

In order to classify this series of cases on an etiological basis I have tabulated the particulars concerning age, symptoms presented, history, antecedent clinical conditions, nature, and results of treatment. These tables are too bulky for reproduction, but after careful study I have grouped the cases according to the principal cause in operation into the eleven etiological groups given below.

Now, in order to assign a single or principal cause to neurasthenia in any given case the greatest care is needed. Not only are many of the causes extremely obscure, rendering repeated examinations necessary, but, as previously pointed out (*loc. cit.*), several causes are nearly always in operation at the same time, and it is often difficult to assign the lead to any one. After careful repeated investigation of the case, two things are necessary: first, *to ascertain whether the supposed causal factor was antecedent in point of time to the neurasthenic symptoms*; and secondly, *to ascertain whether treatment specially directed to the former relieved the latter.* In regard to the first condition the history was subjected to careful scrutiny; many of the cases had been under my observation or that of the medical men with whom they were seen in consultation a considerable time, and eleven of the male patients were themselves medical men. In regard to the second condition I shall have more to say later, but it will be observed that the results of treatment *directed to the suspected cause* of the neurasthenia have been given after the reference number of each case.

The term *cured* indicates that the patient returned to his social or other duties in a normal state of health. In many cases a very long course of treatment was necessary. Some relapsed later.

Relieved indicates that notable relief was observable when the patient was last seen or heard of.

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Unrelieved indicates the converse, and this may have been due either to error of diagnosis, to the intractable nature of the case, or to the unsuitability of the patient's surroundings or the treatment adopted.

Those marked *untreated* did not for some reason adopt the treatment advised.

Unknown indicates that these cases passed from under my care, and in spite of efforts to trace them the result was not revealed; they may or may not have recovered, or become worse.

There were no *deaths*.

M. indicates male and F. female; the numbers are my private case-book reference numbers.

Group I.—Twenty-one cases of neurasthenia due primarily to *intestinal derangement* attended by a greater or less degree of *mucus in the stools*, cases which might, therefore, be classed provisionally as chronic catarrhal colitis, though in the majority of instances this must have been in a very early stage.

Males.	Females.
4, cured.	4*a*, came for colitis, result unknown.
20, relieved.	7*a*, came for colitis, relieved.
31, relieved.	121, cured.
43, relieved.	122, cured.
59, cured.	131, cured.
65, relieved but relapsed later.	
73, unrelieved.	
103, relieved.	
105, result unknown.	
128, cured.	
140, untreated.	
142, cured.	
164, untreated.	
168, relieved.	
177, result unknown.	
182, cured.	

In all of these cases there was a long *antecedent* history of bowel trouble, such as constipation (in the majority) or irregularity of the bowels, intercurrent attacks of diarrhœa, unhealthy, undigested, and frequently highly offensive stools, colicky pains, and other vague abdominal sensations. The presence of more or less mucus, either in chips, flakes, or "casts," was detected in all of these cases, and in some there was a history of it of some duration. But it cannot be affirmed with precision whether the mucus in the stools was or was not really antecedent to the nervous symptoms. Often the mucus was only discovered by accident or admitted after direct questioning, and the precise date of onset of such a symptom must always be a matter of considerable uncertainty.

The treatment adopted in these cases, often extending over a very considerable period of time, was in point of fact that ordinarily employed for mucous colitis, and consisted mainly of dietetic measures, directions as to mastication, systematic purgation, intestinal antisepsis, intestinal lavage, and electricity. Nine were cured, six relieved, one unrelieved, two did not adopt the treatment advised, and in three the sequel of the case is unknown.

Three of these cases may be mentioned as having features of special interest, two of them because they presented skin symptoms which varied proportionately with the neurasthenic symptoms, suggesting that the same toxin which produced the former produced also the latter. I have seen many instances of this association.

Male (20), æt. 32 years, came in August, 1902, with neurasthenic symptoms of over a year's duration. He had a history of constipation and insufficient mastication all his life, with occasional mucus in the stools since about February, 1901, and latterly attacks of diarrhœa, with mucus, specks of blood, and undigested food. A fixed pain and undue resistance in the left hypochondrium made me suspect a band of adhesion, which was partially confirmed by a skiagram. He was operated on by Mr. Leonard Bidwell, and a band was found constricting the splenic flexure of the colon. He made a good recovery and was then gradually relieved by lavage and other intestinal treatment. In August, 1905, Dr. Frood wrote to me that "he is now much improved and in business again, though he still has occasional indigestion, constipation, and mucus in the stools."

Male (4), æt. 45 years, came under treatment March 21st, 1902, with a very long history of bolting of food and constipation all his life; during the last few years occasional attacks of diarrhœa, ending in mucous discharge. Latterly he had had illnesses with neurasthenic symptoms, attended by dermatographia and irritability of the skin, and occasionally large blotches of urticaria. He was cured of both the skin and the nerve symptoms

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after some months of treatment directed to the intestinal canal.

Male (182), æt. 45 years, was sent to me by a medical man in the country for neurasthenic symptoms, associated with recurrent urticaria and pruritus of considerable duration and intractability. After a couple of months' treatment directed to the catarrhal derangement of the bowels, which had been discovered after careful inquiry, he made a complete recovery from both the skin and the nerve symptoms.

Group II.—Eighteen cases of neurasthenia primarily due to *intestinal derangement* (intestinal dyspepsia)—*i. e.* presenting intestinal symptoms identical with the preceding group, associated in some cases with vague gastric symptoms, but unattended as far as could be ascertained with mucus in the stools.

Males.	Females.
9, cured.	10, cured.
18, relieved.	25, cured.
21, cured.	35, relieved.
108, cured.	35*a*, relieved.
129, cured.	36, cured.
156, cured.	68, relieved.
180, cured.	125, relieved.
181, cured.	137, cured.
	141, cured.
	148, relieved.

Treatment on the same lines as in the preceding group, and particularly by intestinal antiseptics and electricity, resulted in cure of both the intestinal and the neurasthenic symptoms in twelve cases and relief in six cases. The following instances may be mentioned.

Male (18), æt. 45 years, came on August 12th, 1902, for neurasthenia associated with inveterate hyperidrosis of the axillæ, preceded by gastro-intestinal derangement, both of the former being relieved by treatment directed to the latter. When I last heard of him the neurasthenia was cured, but the hyperidrosis still troubled him to a less degree. Here, again, the same toxin seemed to affect both the nervous system and the integument.

Male (129), æt. 39 years, came to me in August, 1904, for vague neurasthenic symptoms. I was unable to identify the cause, but in view of the fact that dermatographia was a notable symptom and there was a history of urticaria, I put him on a course of antiseptic intestinal treatment and he made a very rapid recovery.

Male (108), æt. 44 years, came to me in March, 1904, for morbid dreads, panics, and other neurasthenic symptoms. There was a long history of indigestion, unmasticated food, and headaches (which I deemed to be of asthenopic origin). Prolonged treatment directed to the gastro-intestinal condition, extending over upwards of a year, resulted in recovery.

Group III.—Twelve cases of neurasthenia classed as primarily due to *gastric dilatation*:

Males.	Females.
35, cured.	29, result unknown.
39, cured.	39, relieved.
45, cured.	43, cured.
52, relieved.	
67, cured.	
80, result unknown.	
88, cured.	
90, cured.	
100, cured.	

The only true test of gastric dilatation, or muscular weakness (myasthenia), is that the stomach contains food-residues at a time when it ought to be empty, as tested by the gastric aspirator or stomach-tube six or more hours after an ordinary mixed meal. In these circumstances Dr. George Herschell makes three groups of myasthenia—those in which the stomach is found to have emptied itself in six hours after an ordinary mixed meal, those in which it empties itself during the night, and those in which it is not found to be empty even in the morning (which alone proves the existence of true dilatation). It should be mentioned that this test was not applied in these cases, and the diagnosis rested on the percussion signs associated with more or less characteristic gastric symptoms. Under dietetic, electrical, and other measures directed to gastric myasthenia, these symptoms and the nerve symptoms subsided *pari passu* in eight cases and they were relieved in two. In two cases the result was unknown. As examples of this group the following may be mentioned.

Male (35), æt. 43 years, came in November, 1902, for morbid dreads and other neurasthenic symptoms, and he sought my advice preparatory to giving up practice. There was a prolonged history of gastric trouble and evidences of gastric dilatation.

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Measures directed to the gastric condition resulted ultimately in complete restoration.

Male (39), æt. 52 years, came in December, 1902, for attacks of giddiness, unsteadiness in walking, and neurasthenic symptoms of a more definite kind. There was a prolonged history of gastric dyspepsia and evidences of gastric dilatation. When, in the course of a few months, these latter were relieved all his other symptoms disappeared.

Male (90), æt. 59 years, was sent to me in November, 1904, by the late Dr. W. C. Daniel, of Epsom, for curious neurasthenic symptoms, for which the patient had given up work. The leading and most troublesome symptoms were an inability to read and to write consecutive English—an awkward circumstance, for his occupation was that of a journalist. The history and signs pointed to gastric dilatation, and on the adoption of electrical and other treatment for this condition he became quite well, and resumed work in the spring of 1905.

Group IV.—Eighteen cases of neurasthenia, due primarily to dyspepsia and gastric derangements of various kinds without definite evidences of dilatation:

Males.	Females.
10, cured.	1, cured and relapsed.
60, cured.	23, cured.
64, cured.	30, relieved.
70, cured.	52, cured.
178, cured.	54, cured.
	58, unknown.
	89, cured.
	93, cured.
	99, relieved.
	105, relieved.
	106, relieved.
	113, unknown.
	128, cured.

Twelve of these were cured, and four were relieved of the neurasthenic symptoms by the usual treatment for dyspepsia.

As an example the following case may be mentioned.

Male (70), æt. 29 years, came in July, 1903, presenting many interesting neurasthenic symptoms. He was a young doctor, and had treated himself with nerve tonics for many years in vain, and, after talking with me some time he remarked, as though he had made some great discovery, "I am beginning to think that the frequency and severity of my symptoms vary with the state of my stomach." In fact, there was a long history of atonic dyspepsia, and when this was remedied the nerve symptoms entirely disappeared.

Group V.—Three cases of neurasthenia due to various *other abdominal conditions.*

Chronic appendicitis was in operation in 3 cases: male, 57 (result unknown), male, 158 (result unknown), and female, 45 (result unknown).

Dropped kidney existed in 6 female cases (29, 39, 43, 58, 93, 109), but in all of these it appeared to me only to act as a *contributory* cause in association with some other condition. It was not discovered in any of the male cases in this series.

Group VI.—Ten cases of neurasthenia due primarily to certain *septic* or *toxic conditions* (other than those of abdominal origin already mentioned).

Pyorrhœa alveolaris and oral sepsis appeared to be the primary cause in 5 cases:

Males.	Females.
53, cured.	44, relieved.
54, cured.	46, relieved.
	147, relieved.

Oral sepsis was a notable *contributory* cause in 7 cases (male, 31, 90, 128, 177, and female, 4*a*, 58, and 93).

Prostatic blenorrhagia, 2 cases (male, 2 and male, 3), both cured.

Chronic alcoholism, 1 case (female, 19), relieved.

Excessive smoking, 1 case (male, 144), cured.

Antral disease, 1 case (male, 94), cured.

The three cases of appendicitis mentioned in the preceding group might, perhaps, have been included in this group.

This group of cases, when the true cause is detected, forms one of the most curable, and it is not surprising to find that six of these cases were restored to health and four were relieved. As instances the following may be mentioned.

Male (2), æt. 34 years, and male (3), æt. 32 years, both by a curious coincidence came under treatment in March, 1902, for neurasthenic symptoms which appeared to be dependent upon urethritis involving mainly the prostatic portion, for they both got quite well after the latter was cured.

Male (54), æt. 38 years, was brought to me in March, 1903, for numerous neurasthenic symptoms, including a widespread neuro-muscular irritability,

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a symptom which I have observed in a lesser degree in other toxic cases. He had a most severe degree of pyorrhœa alveolaris, and as this became relieved the other symptoms gradually disappeared.

Female (147), æt. 28 years, came to me in June, 1904, for severe neurasthenic symptoms and tachycardia. Electrical and other treatment was adopted without much result, until an aggravated condition of pyorrhœa was discovered. When treatment was directed to this, rapid improvement commenced.

Group VII.—Four cases of neurasthenia primarily due to *fatigue of the nervous system*:

Overwork, male, 83 (cured), male, 150 (unrelieved).

Inveterate masturbation, female, 85 (unknown).

Asthenopia and nervous fatigue, female, 88 (relieved).

Anxiety and domestic worry also acted as a potent contributory cause in many of the cases in the other groups. Asthenopia, which is frequently a consequence of neurasthenia, acted as a *contributory* cause in at least two other cases.

Treatment directed to the primary condition resulted in cure of the nervous symptoms also in one and relief in one. No result was accomplished by treatment in one; the result is unknown in one.

As examples the following cases may be mentioned.

Male (83), æt. 14 years, was sent to me in October, 1903, for nervousness, tremor, hastiness, exaggeration, and a tendency to untruthfulness. He had been overworking and overworrying at school and lately a considerable defect in his vision had been observed. Three months' complete rest, with nerve tonics, cured all his symptoms.

Male (150), æt. 37 years, came to me in March, 1905, with numerous neurasthenic symptoms, which arose, in my belief, mainly from long hours and an unhygienic mode of life. He had gradually become so nervous that he was unable to witness a signature. There was a certain amount of dyspepsia which was secondary in point of time, but treatment directed to this has up to the present afforded no relief.

Group VIII.—Two cases of neurasthenia due to *emotional strain*, male, 85 (cured), and female, 109 (relieved). Treatment directed to the primary condition resulted in cure in one and notable relief in the other case. Male (85), æt. 55 years, came to me in October, 1903, suffering from neurasthenia, with a history of prolonged and intense anxiety. There was also slight gastric atony and cardio-vascular failure. Treatment was, of course, directed to all of these conditions, but in my belief it was chiefly the clearing up of his business worries which led more than anything else to his recovery. He relapsed again two years later under a recurrence of the anxiety, and was cured by the same means.

Group XI. — Various *circulatory disorders* appeared to be in operation as the primary cause in four cases.

Aortic valvular disease and high arterial tension, male, 49 (cured of the nervous symptoms by treatment directed to the cardio-vascular condition).

Cardio-vascular failure, male, 141 (relieved).

High arterial tension of indeterminate origin, male, 37 (relieved).

High arterial tension associated with chronic renal disease, male, 51 (relieved).

In one of these the nerve symptoms were cured, and in three they were notably relieved by measures directed to the circulation.

Male (49), æt. 56 years, came in February, 1903, for marked neurasthenic symptoms. The arterial tension was very high (210 mm.), and there was aortic valvular disease. After a course of Nauheim treatment the tension became lowered and the symptoms disappeared, though the valvular mischief, of course, remained the same.

Male (37), æt. 62 years, was brought to me by his son, a medical man, in December, 1902, for neurasthenia. I could find no physical signs excepting constant high arterial tension varying between 150 and 190 mm., about the causation of which I was never able to satisfy myself. He gradually improved, however, as the blood-pressure became lower under regular purgation and diuretics.

Group X.—Three cases of neurasthenia were found to be dependent on incipient phthisis—female, 130 (result unknown); male (147), mitral disease and incipient phthisis (result unknown); male (157), incipient phthisis (relieved). In one of these cases the neurasthenic symptoms were relieved by attention to the pulmonary condition; the other two passed to the care of others and the

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result is unknown. In two other cases phthisis was a *contributory* cause.

Group XI.—In eight cases of neurasthenia the causes were so complex, and the part which each played so uncertain, that it was impossible to ascribe to any one of them a leading *rôle*.

Males.	Females.
26, cured but relapsed.	67, relieved.
121, unrelieved.	69, relieved.
154, unrelieved.	76, relieved.
	139, unknown.
	154, relieved.

For instance, male (26), a medical man, æt. 46 years, came to me for neurasthenia of considerable duration, associated with gastric derangement, chronic Bright's disease, and overwork, which had gradually supervened in a person the subject of marked nervous heredity. He was cured for a time by measures directed to the first two causes, but he relapsed six months later and committed suicide.

Only one of these was cured (temporarily), four were relieved, two were wholly unrelieved, and the result of one case is unknown.

In two of these some septic condition of the female pelvic organs acted as an important *contributory* cause.

The etiological grouping of these cases is based, as I have said, on the chief cause as diagnosed by a careful review of the case and the revelation of the facts—*first*, that the history of the cause was, as far as could be ascertained, antecedent in point of time to the neurasthenic symptoms; and *secondly*, that treatment directed to the cause relieved or cured the nervous symptoms. Now, in regard to the second of these two conditions, the result of the treatment adopted in each case has been mentioned after the reference numbers of the cases in the above groups, and a summary of these results is now given in a tabular form below. The meanings of the terms "relieved," etc., at the head of the columns have been previously defined. This table is very instructive from the point of view of prognosis, and effectually disposes of the idea that neurasthenia is an incurable disease. Eighty-three out of 103 (80·5 per cent.) were relieved or recovered. Very prolonged treatment was often necessary.

Table showing the Results of Treatment in the several Etiological Groups in 103 *Private Cases of Neurasthenia.*

Etiological groups.	Cured.	Relieved.	Unrelieved.	Untreated.	Unknown.	Total cases.
I. Intestinal cases with mucus	9	6	1	2	3	21
II. Intestinal cases without mucus	12	6	—	—	—	18
III. Gastric myasthenia	8	2	—	—	2	12
IV. Dyspepsia of various kinds	12	4	—	—	2	18
V. Other abdominal conditions	—	—	—	—	3	3
VI. Other toxic and septic conditions	6	4	—	—	—	10
VII. Nervous fatigue	1	1	1	—	1	4
VIII. Emotional and traumatic shock	1	1	—	—	—	2
IX. Circulatory troubles	1	3	—	—	—	4
X. Phthisis	—	1	—	—	2	3
XI. Complex and indeterminate	1	4	2	—	1	8
Totals	51	32	4	2	14	103

However, we are not now concerned with the prognosis but the pathology of neurasthenia, and the point is this: if the effects of treatment were successful, these go far to confirm the etiological grouping, but not otherwise. In order, therefore, to obviate etiological fallacies as far as possible I propose to base my conclusions on the *eighty-three cases* in the first and second columns of the above table, in which the diagnosis of the cause was confirmed by the results of treatment directed to that cause, these cases being either cured or notably relieved thereby.

Analysis of the Causes of 83 *Cases of Neurasthenia in which the Diagnosis of the Cause was confirmed by the Results of Treatment.*

	Cases.	Per cent.
Intestinal derangement, with or without mucus	33	*39·9*
Gastric dilatation and various other kinds of gastric disorder	26	*31·3*
Other septic and toxic conditions	10	*12·0*
Fatigue	2	*2·4*
Emotional and traumatic	2	*2·4*
Circulatory and lung troubles (? malnutrition)	5	*6·0*
Complex causes	5	*6·0*
	83	

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In passing it may be mentioned that in the series of hospital cases which I published in 1898 * the chief cause in operation was found to be "gastric disorder"—74 out of 157 cases of neurasthenia (47·1 per cent.) If, therefore, it be remembered that a number of intestinal and gastro-intestinal conditions were probably included among these hospital cases, owing to the difficulty of analysing the precise nature of the alimentary disturbance, the percentage of gastric disorder in the private cases (31·3 per cent.) and that in the hospital cases bear a fairly close resemblance.

But undoubtedly the most striking and important circumstance brought out by an examination of this series of private cases is the great preponderance among the causal conditions of neurasthenia of intestinal (33 cases) and gastric derangements of various kinds (26 cases), which together amounted to 59 out of the 83 cases which we are now examining (*i. e.* 71 per cent.). This preponderance confirms and extends the conclusions I have previously arrived at. In both series there is a notable predominance of the same kind of cause. In these private cases, however, much greater accuracy both in the detection of gastric and intestinal disorders and in the analysis of the precise nature of the alimentary disturbance is attainable for reasons previously mentioned.

The occurrence of so many cases of neurasthenia in which, upon careful inquiry, evidences of some antecedent disturbance of the alimentary canal was found to have existed, and in which the relief of the alimentary disturbance was attended by an amelioriation of the nervous derangement, merits careful consideration, and, if possible, explanation. I am the more concerned to point out this causal relation again because it has not, as far as I am aware, been pointed out in writings on the subject, and it appears to me that both the various gastro-intestinal conditions here referred to and their true relation to neurasthenia are very frequently overlooked. All of these fifty-nine cases sought advice, not for any abdominal condition, but for the neurasthenia. In some cases the abdominal derangement had not been detected, or, if detected, had been regarded as of minor importance. In others the gastric or intestinal symptoms were regarded as part of or *as a consequence* rather than the cause of the neurasthenia. Many of these patients had wandered from one "cure" to another, or had tried every conceivable form of dietary, or taken nerve tonics *ad nauseam* without getting relief until adequate measures were directed to the gastro-intestinal condition. This oversight of relationship is not surprising when one remembers the prolonged and insidious character of the earlier symptoms of most intestinal conditions.

That the gastric and intestinal symptoms were not a result, or an integral part, of the neurasthenia in this series of cases was evidenced by a careful study of the history of each of these patients, by the failure of treatment directed at first in some cases to the neurasthenia alone, and by the fact that treatment directed to the abdominal condition always cured or relieved both the gastro-intestinal and the nervous symptoms in the 59 cases under consideration. This question has already been fully discussed on a previous occasion, and it is quite clear that the neurasthenia is the result of the gastric or intestinal derangement. This is the most salient fact brought out by this series of cases.

How, then, and in what manner does gastro-intestinal derangement produce neurasthenia? Is it a question simply of malnutrition—*i. e.* insufficient or perverted nutrition of the nervous system? I think not, else why should not the other physiological systems suffer more? Occasionally one finds that when these patients have at last come for relief they are losing weight, but this is not the rule, and even in these cases the nervous symptoms had generally been going on long before the loss of weight began. In 1899 (*loc. cit.*) I showed, contrary to the views then prevailing, that gastro-intestinal derangement produces neurasthenia by means of an auto-intoxication of the nervous system, and this conclusion is supported by an examination of the fresh data now submitted. That gastro-intestinal disorders are attended by fermentation and are capable of producing an auto-intoxication we know from various considerations. A critical examination of the intestinal cases in Groups I and II, which differ only in the presence (21 cases) or absence (18 cases) of fæcal mucus, shows that even in the former the amount of mucus was, with very few exceptions, never great, was always intermittent, and might disappear altogether for a considerable time. If, then, the cases in Group I could be classed as colitis, the

* *Loc. cit.*, 1st edition, p. 69, *et seq.*

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majority of them belonged only to the prolonged incipient stage of that malady—one might almost say to an ante-colitis stage when only "intestinal derangement" and constipation were constantly in operation and actual catarrh of the bowel and diarrhœa only present from time to time.

In these earliest phases of the bowel troubles which lead to colitis, diarrhœa and fæcal mucus are generally absent, but we get all the evidences of chronic intestinal fermentation—flatus (gastric and rectal), borborygmi, colicky pains from wind passing along the bowel, irregularity of the bowels, undigested and offensive stools, excess of *Bacillus coli* in the stools, and mucus (when present) preceded or associated with constipation rather than diarrhœa. Now, these are just the symptoms which existed in the thirty-three cases in Groups I and II, and these were really cases of intestinal fermentation, attended in a certain number by more or less mucus in the stools.

Some authors state that mucous colitis is always attended by nervous symptoms, though others deny this. My experience has led me to believe that in the first, the prolonged, insidious (the ante-colitis) stage, which may go on for years in a more or less intermittent fashion, neurasthenic symptoms always arise, and further, that when the colitis reaches a later and unmistakable stage, where the amount of mucus in the stools forms perhaps the most striking and pathognomonic feature of the case, nervous symptoms disappear or become less prominent. This, I believe, accounts for the fact that many bath-physicians of great experience in the treatment of *advanced* colitis deny the constant presence of neurasthenic symptoms in cases of colitis.* This is probably true at the stage when the patient is sent to the bath station for intestinal treatment.

Next in number amongst the abdominal causes of neurasthenia come twelve cases which I have grouped under "Gastric Myasthenia and Dilatation" (to which the remarks previously made as to the unsuspected existence and relationship to the neurasthenia equally apply), and these are closely followed by eighteen cases of various forms of dyspepsia. In both of these groups we know that fermentation occurs, particularly in the former. In both, lithuria and other evidences of perverted metabolism are constant.

* *E.g.* Dr. Bottentuit of Plombières-les-Bains. 'British Medical Journal,' June 27th, 1903.

Nothing was more constant in these fifty-nine cases than the ashy complexion so characteristic in toxæmia, and a considerable number of the cases in the four groups under discussion presented dermatographia, urticaria, twitching of the limbs, and other evidences of toxæmia. And, finally, antifermentative measures, both gastric and intestinal, were undoubtedly the most successful means of curing or ameliorating both the morbid condition of the alimentary canal and the neurasthenia in all of these fifty-nine cases.

As to the nature of the toxin which arises in the gastro-intestinal cases, considerable doubt must exist. It may be one of the chemical products of intestinal fermentation, or it may be microbic : in the present state of our knowledge it is not possible to decide which. Many microbes are found in the intestines, but suspicion is at once fixed on the *Bacillus coli communis*, which although it is a very constant inhabitant of the large intestine, is greatly increased when the mucous membrane is unhealthy or fermentative processes are going on in the intestines. At one time it was regarded as non-pathogenic, but we are gradually learning that the *Bacillus coli* may have far-reaching pathogenic effects, and some observers have attributed to it specially disastrous effects on the nervous system. I have seen more than one case where a genito-urinary infection by this microbe was attended by very serious nervous symptoms. It seems certain however, that the gastro-intestinal canal is the laboratory where a toxin is manufactured which by its effects on the nervous system (predisposed perhaps by fatigue, heredity, or some other contributory cause) produces neurasthenia.

There may be another explanation of the causation of neurasthenia by intestinal disorder We know that all gross intestinal lesions are attended by a totally disproportionate amount of prostration, and Sir Lauder Brunton has very kindly indicated to me some experiments by Ludwig and Dolgiel in 1867 * which showed that a mechanical stimulus applied to the intestines greatly accelerated the flow of blood through the carotids. Dr. Leonard Hill † more recently has

* "Sitzungeberichte der math.-phys. Classe der K. S.," 'Gesells. d. Wissensch.,' Band xix, p. 257, and Ludwig's 'Arbeiten' 1867, p. 253.

† 'The Physiology and Pathology of the Cerebral Circulation.'

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shown something of the same kind, namely that the blood supply to the brain varies inversely with that in the abdomen. In this way a direct or reflex nervous influence might be invoked through the sympathetic nerves terminating in the intestines. But in view of the generalised nature of the nervous derangement in neurasthenia, which is not, as we have seen, limited to the brain, it seems much more probable that the essential pathological change is a widespread toxæmic or hæmic change of some kind which damages all the nervous structures. The cæcum is a cloaca in which fermentation and bacterial changes are continually going on, as Mr. Arbuthnot Lane and others have pointed out.

Passing to a review of all the possible causes of neurasthenia, all the causal agents of neurasthenia may, in my belief, be brought under one of four pathologcal factors — Toxæmia of the nervous system, Fatigue of the nervous system, Emotional and Traumatic causes, or Malnutrition of the nervous system. The series of 103 cases we are now considering may, therefore, be thus classified on a pathological basis as follows:

Class A.—Toxæmia of the nervous system: Groups I and II, intestinal derangement, with or without mucus—49 cases; Groups III and IV, gastric myasthenia and dilatation and various forms of dyspepsia—30 cases; Group V, other abdominal conditions—3 cases; and Group VI, other toxic and septic conditions—10 cases. Total, 82 cases.

Class B.—Fatigue of the nervous system: Group VII, overwork etc.—4 cases.

Class C.—Emotional or *traumatic* shock: Group VIII—2 cases.

Class D.—Malnutrition of the nervous system: Group IX, circulatory troubles, and Group X, phthisis (provisionally classified here)—7 cases.

Complex causes unclassifiable: Group XI—8 cases. Grand total, 103 cases.

This series of 103 cases is sufficient to draw fairly definite conclusions. It follows, therefore, that in the largest proportion of cases (83·2 per cent. of the 83 cases verified by the effects of treatment, and 79·6 per cent. of all the 103 cases) the cause of the neurasthenia is an auto-intoxication specially affecting the nervous system and so producing symptoms of nervous exhaustion, instability, or irritability. The nervous system may also be damaged by other means; it may be rendered unduly susceptible by overwork, worry, heredity, and what not. There is a compound cause in operation in nearly every case. Moreover, worry and anxiety about trifles, and restlessness, which are the symptoms or consequences of nervous exhaustion, may of themselves produce further nervous exhaustion. But to break this vicious circle it is necessary to identify and attack the primary cause.

Before concluding I should like to take one step still further back in the etiology of neurasthenia and inquire what is the causation of the gastro-intestinal sepsis and auto-intoxication which are such potent factors in the pathology of this disorder. *Rich foods* and dietetic errors played an important part in some of the wealthier patients. A long history of *constipation* (which was present in 17 out of the 21 cases in Group I) undoubtedly tended to the production of the intestinal sepsis. But there is no doubt in my mind that the leading *rôle* was taken by *insufficient mastication* of food. In no fewer than 19 out of the 21 cases forming Group I (those in whom mucus was at some time discovered in the stools) there was a distinct history extending over many years of insufficient mastication, and a similar history was revealed in many cases in the other groups, I to IV. In all four groups many of the patients when first seen had very bad teeth (even apart from pyorrhœa alveolaris), and it was not until these had been remedied and they had been taught to use their teeth that their condition really improved. Others, clerks and business men and women, volunteered or admitted that by the nature of their work their meals were extremely hurried. Others, higher in the social scale, admitted or complained that the carrying on of interesting conversations at meal-times was inconsistent with adequate mastication. Many of these neurasthenic subjects are persons of very considerable intelligence and brilliancy, who, therefore, take a leading part in society, and are much in request as diners out. Many of them belong to the nervous type of individuals who take life hurriedly, including meals. Among my hospital out-patients the same conditions of hurried meals and insufficient mastication obtained. The presence of bad teeth and a history of bolting food is revealed with monotonous iteration among the procession of neurasthenics which passes through the out-patients' room.

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The conclusion to be drawn from the above is that although the rapidity of life in our modern civilisation acts as an important factor in the causation of neurasthenia, the habitual bolting of food and the insufficiency of mastication, coupled with the tendency to decay of the teeth at an earlier age than in previous generations, are even more important.

Verily "the pace we live at" is deleterious, by the wear and tear of the nervous system; but it is the pace at which we eat, together with the early decay of our teeth, which are mainly responsible for the increase of neurasthenia in modern times.

Prognosis.

Some valuable lessons as to prognosis may be drawn from this series of 103 private cases, where treatment could, in most instances, be more thoroughly and adequately adopted. They were all seen for the first time between February 27th, 1902, and August 18th, 1905. Many of them were under treatment for a considerable time, and I have traced the result in all those where it was possible. In 15 cases the result is unknown, either because the patients passed from under my care or could not be traced; they may or may not have been cured or relieved. In 3 the treatment advised was not carried out. Of the remaining 84 cases in which the treatment advised was carried out and the result known the neurasthenia was cured in 46 (54 per cent.), notably relieved in 33 (40 per cent.), and failed either to cure or relieve in 5 cases (6 per cent.). Two of these 46 cases relapsed some time later to my knowledge, and some of the others may have relapsed or may hereafter relapse, without my knowledge, but they all returned to work and became, to all intents and purposes, well. On the other hand, of the 33 cases classed as notably relieved, some may have subsequently recovered; in some complete relief was prevented by their environment, in others by the confirmed establishment of the disease, and in others by the incurability of the cause.

The recovery of 46 cases (54 per cent.) and the relief of 33 (40 per cent.) is a sufficient refutation of the statement which some make that neurasthenia is essentially an incurable condition. The more I see of neurasthenia the more I am persuaded, first, that neurasthenia is capable of cure or amelioration in a large proportion of instances, and secondly, that its curability depends upon two chief factors—(1) the discovery of its cause, and (2) the possibility of applying adequate means for the removal of the cause. It is often extremely difficult to detect the chief cause in operation. The most thorough and painstaking investigation, based on a wide knowledge of general medicine, is called for, as a list of the etiological groups above mentioned will show. For success a case must be repeatedly investigated, all the various possible causes considered, and their relative importance carefully weighed.

But if the discovery of the cause is difficult the possibility of adopting adequate means for its remedy is often still more so. This may mean a prolonged course of treatment, which alters the whole mode of life, which may be quite beyond the means of the ordinary individual, who has perhaps already tried many "cures" and lost his faith in "doctors," who has, in the process, established, through a long course of time, a confirmed and vicious condition of the nervous system, and who is apt to question the necessity of abandoning old habits for the doubtful cure of a disease which, however chronic and however inconvenient and incapacitating, is regarded by him as non-fatal.

There are, of course, other factors in the prognosis of neurasthenia which have to be considered. The previous duration is one of these, and I am accustomed to tell the patient that the treatment required will probably need as many months to relieve as the years through which it has lasted. But there are certainly some cases which, by their long duration, it is impossible to cure even when the cause is removable. The question of age is also a factor in prognosis, and undoubtedly after fifty it is often impossible to completely restore health, especially where the malady is of long duration.

A history of nervous heredity also adds greatly to the gravity of the prognosis and the difficulty of cure, particularly in cerebral neurasthenia. In such cases the delusions and other mental symptoms are very apt to become persistent, and the patient gradually drifts into chronic insanity. Changing delusions are not uncommon even in favourable cases, but the persistence of any one delusion, and especially a delusion of persecution or being followed, is generally grave, and so also are hallucinations of hearing and sight.

June 4th, 1906.

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A CLINICAL LECTURE

ON

THREE CASES OF TUMOUR IN THE NECK.

By SIR WILLIAM BENNETT, K.C.V.O., F.R.C.S.,

Consulting Surgeon to St. George's Hospital; Surgeon to the Seamen's Hospital, London Clinical School of Medicine.

GENTLEMEN,—The cases which we have before us to-day form an interesting series, especially in relation to the value of probability in rational diagnosis, a factor which seems to me to be in danger of being under-estimated in these days of searching for the rare and unusual. The remarks which follow may be taken as a sequel to those on the predominant importance of the local aspects of disease in diagnosis which appeared in the CLINICAL JOURNAL of October 18th, 1905.

I. A FLUCTUATING SUBMAXILLARY TUMOUR.

Here is a girl, 17 years old, of a somewhat delicate type who has a fluctuating, softish swelling under the angle of the jaw on the left side; the overlying skin is of a natural colour, and there is only slight tenderness; the tumour is not bound down to any material extent, but it cannot be said to be freely movable. The situation is a common one for inflamed and suppurating glands in connection with infective conditions of the throat, especially in the neighbourhood of the tonsil. The patient tells us that three years ago, following upon sore throat, a lump was noticed; at first it was soft and tender, but it appears never to have been very painful. These are two points to be carefully borne in mind in connection with the diagnosis. At the beginning of the present year some adenoids were removed after which a rapid increase in the tumour took place, so that there is good evidence

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to show that she has had, within the time that this lump has been in existence, a sufficient source of irritation in the throat to account for the occurrence of glandular infection in this situation. Naturally your mind is at once directed to the probability of the case being a glandular abscess, and at first you would hardly be inclined to think of anything else beyond an abscess, secondary to some infective process about the throat. A hasty diagnosis is, however, always unwise, and there are two or three points about the case which may lead you to hesitate before you commit yourself finally. First of all, you will notice that the swelling is very soft, it is very distinctly oval, its edges are very clearly defined, and, what is perhaps most important, so far as you can feel, there are no other small glands involved round about it. It is very common in glandular disease following upon throat trouble to find round about the main lesion other small glands which become involved secondarily to the changes which have taken place in the glands originally concerned. Given a fluid swelling of a chronic kind under the angle of the jaw, in a girl who has been subject to certain conditions such as cause from time to time a "sore throat," for example adenoids, you would naturally conclude that the case is probably one of chronic abscess in connection with gland infection, and probably you would be right. But before committing yourself to that diagnosis you must consider whether it is possible that the condition may be something different; in other words, you must as far as you can eliminate certain possibilities before you accept probabilities without reserve. If this is not abscess following upon a diseased gland, the alternative would be a dermoid of some kind, and it has been suggested that it may be in connection with a defective branchial cleft, but this is a rare condition, and it would require strong evidence to lead you to accept this last alternative against the probability of glandular abscess. The points justifying the consideration of a dermoid being the condition are, the extreme fluidity of the contents of the cyst, the flaccidity of the cyst itself, and the entirely healthy condition of the overlying skin, which can be pinched up quite free of the tumour. Speaking generally, a glandular abscess is somewhat tense, and very frequently its contents are not so fluid as they seem to be here, because in many of these cases tuberculosis exists, and the contents of the abscess are not always merely pus, but consist of a mixture of broken-down granular tissue and tuberculous foci, which give to the touch the suggestion that the tumour contains jelly-like material rather than a watery fluid. In spite of all that, I think if you were faced with this case at an examination you should pin your faith on its being in all probability one of chronic abscess. The swelling is very fluid, as I have said, it is very well defined, and no other glands involved can be felt about it. Supposing it is chronic abscess connected with the glands round about the tonsil in consequence of some infective process which has been going on inside the throat, the tumour which you see may be a circumscribed collection of pus which has gradually escaped from a gland which is deeper down; a common condition, and one of much importance in the matter of treatment, because you can readily see that the lesion which is apparent is under such circumstances superficial and only a secondary result of the disease of the gland; hence, assuming for the moment that this is an abscess secondary to glandular disease, a mere opening or scraping of the abscess would be useless unless the diseased gland itself were at the same time removed. In other words, the tumour which you feel is probably not the gland itself; but is due to the escape of the results of suppuration from the interior of the gland. Supposing you were to incise the swelling, assuming again that it is a glandular abscess, you would expose a well-defined and clean abscess cavity, which with a little scraping you could clear out apparently quite easily. But upon further careful examination you would almost certainly find deep down at the bottom a small hole, through which a probe might pass, leading through the deep fascia into the middle of the gland beneath, from which the material forming the abscess originally escaped. It is very important to bear in mind, in connection with all these glandular abscesses about the neck, that, speaking generally, whenever you get a large, well-defined abscess of this kind what is felt is not the originally affected structure, but only what has come from it, and that you will altogether fail in your treatment of such a case unless you eradicate the deep disease of which the superficial portion is a secondary consequence only. I have seen on more than one occasion abscesses of this type

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opened and scraped out without further investigation to the entire satisfaction of the operator apparently—a satisfaction which is apt to be much modified by the rapid recurrence of the abscess, because the real seat of the trouble has not been dealt with. The tendency of pus in the substance of a gland to escape through the capsule into the soft parts around before the whole gland has been destroyed by suppuration increases with the age of the patient. In infants and very young children the destructive process is so acute that the whole gland is often completely destroyed and rapidly converted into an abscess even in tuberculous disease, as distinct from ordinary infective adenitis. The result is that in infants and the very young the mere opening of the abscess of this type is frequently all that is necessary for a cure, because the diseased gland has been destroyed in the process of suppuration; at the same time, even in such cases, care should be taken to see that there is no evidence of an underlying mass of disease. A very fair guide is the situation of the abscess; if this is under the deep fascia, it may be considered to be complete in itself; if superficial to the deep fascia, it is probable that a partially destroyed gland is lying underneath, and a most careful investigation should be made in that direction.

II. A Tumour of the Thyroid.

Here is another patient who has a tumour in the neck. The tumour is just above the episternal notch; its shape is oval, it is smooth, it moves about from side to side, and you can raise it and depress it. When the patient swallows it rises and falls with the larynx. It is clear therefore that it must be a tumour of some sort connected with the thyroid gland. The history shows that the swelling in the neck has existed for at least six years, and that it has been increasing very slowly until recently, when it has rather rapidly grown. Generally it has been noticed to become somewhat larger at the catamenial periods. There has been no difficulty in breathing, but headache and giddiness occasionally occur. There is no cough and no change in the voice. Further, there has been no difficulty in swallowing; it is therefore obvious that whatever the condition may be it has not given rise to any great trouble. Note for a moment the patient's aspect; she is inclined to have some capillary injections of the cheeks, and her face generally looks congested. I think it may also be said that her eyes show a tendency to protrusion, although there is nothing like exophthalmos. The pulse is rather quick, but that may be from the temporary excitement caused by having so many critical people around her; there is, however, nothing about it which suggests anything especially wrong with her cardiac arrangements. I expect she suffers from shortness of breath during exertion, and she now tells us that this is so. She was sent here with a statement that the condition of her heart and circulatory system was so bad that under no circumstances should she be allowed to take an anæsthetic. If the condition of her circulatory system is affected to such a degree it is clear she can derive little benefit from being here, because the only thing we can do with the tumour is, if it seems worth while, to remove it. If she is not in a condition to take an anæsthetic, as has been suggested, I do not think we could reasonably set about any radical proceeding, and we must leave her as she is. But as a matter of fact I think you may dismiss the question of the special danger of the anæsthetic in connection with the condition of her heart and circulation altogether, provided that the administrator is a skilful person. I do not know what the reason is, but there seems to be a pretty strong idea with some people that conditions of the circulatory system often exist which may be a positive bar to the administration of anæsthetics. I have seen a great deal of the administration of anæsthetics as an operator, and years ago I administered anæsthetics some thousands of times, but I have never seen a patient whose circulatory and heart condition, in the absence of pulmonary complication, was such as to be a bar to the taking of a properly administered anæsthetic. So you may safely set aside any fear with regard to the anæsthetic; of this I am sure from the appearance of the patient and from the fact that she has had no pulmonary complication. It is true that she has a pulse which is a little quick and irregular at times, but you know that the association of a quick, rather erratic pulse, sometimes even a very quick pulse, is not at all rare in tumours and other conditions affecting the thyroid gland, independently of what is commonly called exophthalmic goitre. So we need not hesitate to give this particular patient an

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anæsthetic. We can therefore now consider whether it is a proper thing to advise the removal of the tumour or not. There is a striking injection of the capillaries about the face which is unnatural and may be connected with the thyroid condition. The tumour is increasing in size; there is no difficulty in breathing nor of swallowing, either in the upright or flat position. Many people who are the subjects of thyroid tumours suffer from dyspnœa when they lie flat on the back, though in the upright position they may be quite free from it when the tumour is of moderate size, especially if it is situated well away from the middle line; the position of the tumour thus has an important relation to its tendency to cause trouble early in its growth—the nearer the middle line the sooner as a rule do symptoms arise. It may be well to remind you of a peculiar characteristic in the difficulty of swallowing which occurs in these cases. You may perhaps remember that in discussing a case of malignant disease of the gullet a week or so ago in the Fitzwilliam Ward we noticed how vividly the patient described the manner in which he was gradually driven by the increase of his disease to give up all solid food, as he found by degrees that he could swallow nothing but liquids. His trouble began so far as he knew with the difficulty of swallowing solids. In the dysphagia connected with thyroid tumours, etc., the trouble generally commences with difficulty in swallowing liquids, solids being taken quite well.

These points are of some clinical importance, because it frequently happens that patients seek advice on account merely of difficulty of swallowing in cases of this kind, the condition in the thyroid being too small to attract attention, or if it has been noticed the difficulty in swallowing has not been associated with it in the mind of the patient. The first question you should ask therefore when consulted in this way is this, Is the difficulty arising in attempts at swallowing caused by solid food or by fluids? If the difficulty is connected with solid food, the dysphagia should be due to disease of the gullet; if it is connected with fluids, it points to some abnormality of the thyroid or larynx.

With regard to the patient before us, the tumour is increasing. She has had no symptoms of discomfort as yet, but they will come when the tumour gets larger. It should be dealt with now if it is to be dealt with to the best advantage. The tumour is a cystic adenoma of the thyroid, probably of a soft-solid consistence. There are two principal forms of these bronchoceles, cystic and solid, and I think this is one of the soft-solid kind. The mass is very distinctly encapsuled, and if the actual surface of it is exposed it shells out without great difficulty, something in the way that a sebaceous cyst comes from its capsule in the skin, although less easily. For its removal no extensive operation will be required. The skin incision over the tumour should be free; it may follow the line of the anterior margin of the sterno-mastoid, it may be crescentic (transversely), or it may be angular, according to the taste of the operator. I always use the oblique incision, enlarging it by an angular extension from the lower end if the size of the tumour render enlargement necessary. This wound I have found to form a less unsightly scar than that following the other incision, and I have seen nothing of the puckered scarring after the oblique incision to which reference is sometimes made. It is said, on the other hand, by some people that the transverse or semilunar wound heals better and that the resulting scar shows less. But there is one objection to that incision, especially in girls and young women, and it is to them sometimes a matter of much consequence. The transverse form of scar is suggestive of a self-inflicted wound, and may be confused by inexperienced people with the scar resulting from cut-throat—not the most becoming scar with which to pass through life. This may be held to be merely a captious objection, but in a case which came under my notice it was a matter of vital importance and the end was deplorable. The tumour having been exposed, I am content with shelling it out, an operation of extreme simplicity, and when properly performed one which gives rise to little or no loss of blood. In cases in which two or three tumours exist I remove them in the same way, if there are more I remove the thyroid lobe affected. In enucleating these tumours the one point of supreme importance is to be sure that the actual surface of the tumour is exposed; the actual surface having been in part laid bare, the tumour can be enucleated with the finger or instrument—I prefer the finger—without much difficulty. If any attempt is made at enucleation before the yellowish-white surface of the tumour be exposed, the substance of the

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thyroid itself is opened up and furious bleeding may follow. After the tumour has been properly enucleated some welling up of bright blood may follow, but if you will treat this merely by stuffing a sponge into the cavity, you will find that in a very short time the bleeding will cease. I have never seen bleeding continue under those circumstances in a properly performed operation. If, on the other hand, you have not sufficient confidence in the tendency of the bleeding to stop spontaneously and you begin to plunge about with pressure forceps trying to pick up the structures in the deep part of the cavity from which the blood comes, you will almost invariably tear and lay open the thyroid tissue, thus converting a condition which was previously simple into a serious one, in which hæmorrhage may prove disastrous, and may lead to your having to remove half of the thyroid gland in order to stop the bleeding resulting from the mismanagement of this comparatively trivial operation. Several years ago I expressed the same views with regard to this operation in a lecture published in the CLINICAL JOURNAL, and a surgeon of repute took me somewhat to task in a communication published later in the same journal, suggesting that my lack of experience accounted for my views, which he adversely criticised. My experience then was not small, now it is much larger, and I repeat, as my deliberate conviction, that the enucleation operation is not associated with any danger from hæmorrhage if it is properly performed.

After the removal of a tumour like this, no matter how nicely the operation may have been done, and in spite of the most elaborate precautions, you must not be surprised if during two or three days subsequently a high temperature develops. But you need not on that account rush to the conclusion that there is septic infection, because the high temperature and very quick pulse—perhaps running as high as 140 or 160 beats to the minute—sometimes occurs after operations of this kind, not on account of septic infection, but in consequence of the interference with the substance of the thyroid in the course of the operation. The thyroid body is a very mysterious one, and no doubt has an extraordinary relation to the animal economy. Interference with it by operation may, as I have already said, lead to the development of a high temperature and intensely quick pulse, quite independent of septic trouble. This is a most important point to bear in mind, because if you make the mistake of attributing these symptoms to septic infection, the first thing you would naturally do would be to open up the wound to allow of the escape of any retained septic material. If you pursued that course you would probably be doing the worst possible thing for your patient, because you might again interfere with the thyroid and only intensify the condition which you had in your mind to mitigate. Therefore bear in mind in cases of operation on the thyroid gland, that reactionary fever and rapid pulse need have nothing to do with septic infection. Should they occur a careful watching of the patient through the crisis—and sometimes it is a heavy crisis—usually ends in recovery. All other things being equal, you may consider that the more the thyroid tissue has been opened up in the operation, the more likely is this peculiar reaction of high fever and quick pulse to follow. That is one of the reasons which led some people to advocate removal of half the thyroid in preference to the partial operation; it is said that in the removal of half the thyroid there is less liability to these reactionary symptoms than when the tumour is shelled out from the substance of the gland. That depends on how the operation is done, because in removing part of the thyroid you have to divide the isthmus, and sometimes that is very thin and sometimes very thick. If in the enucleation operation you avoid tearing up the thyroid by taking care to get into the capsule of the tumour before you disturb it, you need not fear the reactionary conditions which I have mentioned. Now, the tumour in this patient will never recur if it is taken out; but sometimes, although there may appear to be only one tumour of this kind, there may be lying about in some portion of the gland one or more other small tumours. So if by chance another tumour should develop later on it would not be a recurrence of the present one, but it would be due to the growth of another small tumour of a harmless kind. This is an innocent tumour, there is no suggestion of malignancy about it. Malignant disease of the thyroid is not so rare as is commonly supposed, but it is not so common as to give you an opportunity of seeing it often. The prognosis in this case is perfectly good and the operation is not at all severe; the tumour should therefore be removed,

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and as you have heard, the patient agrees to this course.

III. An Apparently Fluctuating Tumour over the Parotid Gland.

The next case is an excellent one for comparison with that which we have just been considering, and it well illustrates how necessary it is to be careful in arriving at a definite conclusion as to the nature of a case, however obvious the local condition may seem to be. I first saw this girl outside the hospital about a week ago, and was told that for six or seven weeks she had been troubled with a lump under the ear in the situation which you see now, over the parotid gland. Her illness began with a "cold." But, although she had a little stiffness in her neck, she had never noticed any sore throat. Of course upon seeing a tumour of this kind below the lobule of the ear over the parotid gland, the first thing which naturally suggests itself is the possibility of its being one of those soft cartilaginous tumours which are commonly met with in that region. But you should never commit yourself to a diagnosis of anything about this region in the way of a swelling until you have carefully examined the inside of the patient's mouth. On the occasion of my first seeing this patient the tumour over the parotid gland was very hard, almost as hard as a marble, and on looking inside her mouth there was, as there is now, on the left side, an abnormally large tonsil, reaching as high as the junction of the hard and soft palates, on the inner edge of which there was an excavated ulcer which would admit the tip of my little finger. In those circumstances, seeing that the other tonsil was absolutely sound and normal, the first thing which came into one's mind was, the possibility of malignant disease of the tonsil, with secondary infection of the glands externally. Then came the question, Could this be tuberculous disease of the tonsil with the corresponding affection of the glands in the usual situation? Those were the two possibilities which presented themselves to me at that time. Reverting for a moment to the point of which I spoke at the commencement of these observations, viz. the value of probability in diagnosis, you will find that although it may deceive you at times, probability will generally be a good guide. Now, the tonsillar mischief here is practically painless; the patient was unconscious of her tonsil being affected, and, as I have said, she has had no sensation of sore throat; bearing those facts in mind in the case of a girl of this type, it is thinkable that the case may be one of simple chronic hypertrophy of the tonsil, with ulceration and consequent septic infection of the external glands, and not one of acute or grave pathological change, such as sarcoma, carcinoma, or tubercle; on the whole, however, the malignant view seems the more likely to be correct.

I therefore came to the conclusion that sarcoma was the condition. I consequently sent her to the hospital for observation and operation if such seemed feasible. In the week which has passed since I saw her a great change has come over the case. The condition of the tonsil is unchanged, but the tumour over the parotid has doubled its size, is soft and apparently fluctuating; in fact, it looks uncommonly like an abscess. Unhappily, this development of an apparent abscess does not negative the possibility of the tonsil condition being sarcoma or even carcinoma; for the glands occurring in connection with ulcerative sarcoma may be merely infected by septic organisms, and the glands secondary to carcinoma in mucous membranes are sometimes nearly fluid. So my original diagnosis of malignant disease is not materially shaken by this change in the external swelling. Under those circumstances, the next thing will, of course, be to ascertain whether the contents of the swelling are pus or not, and if they prove to be mere pus the rational thing will be to clear out the large tonsil, but I fear the contents will not be pus. The fact of the tonsil on the other side being absolutely normal is somewhat against the condition being a septic trouble consequent upon chronic hypertrophy of the tonsil.*

A point which the patient mentioned in connection with her history, which I omitted to mention, is of interest; she stated that the tumour varied in size from time to time, being sometimes small whilst at other times it was large. What is the explanation of this? It is a matter of common knowledge that glands the seat of chronic tuberculosis do vary in bulk from time to time, or at all

* Upon opening the supra-parotid swelling its contents proved to be softish material clearly secondary to carcinoma of the tonsil. The case was quite beyond the scope of justifiable interference by operation.

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events are said by patients to do so; it is not always easy to explain the reason of this variation, but it is sometimes a fact. In this particular case the explanation of the apparent variation in size is not difficult, seeing that the tumour was intimately connected with the parotid gland. The salivary glands sometimes vary, it is needless to say, in tension, etc., a good deal, especially in hungry people at the prospect of a meal (increase of tension in the gland would give a sensation of increase in size in a tumour immediately overlying it although no actual increase in size may occur); if the tumour were an outgrowth or a part of the salivary gland, a considerable real variation of size may occur for the same reason from time to time. My main reason for mentioning this matter is to impress upon you the necessity in surgical diagnosis of taking nothing for granted, but to make a point of withholding your judgment until when a patient makes a statement of this kind you have had an opportunity of verifying it personally. Nothing is so deceptive as the sensations of a patient with regard to the alteration in the size of a part, a fact which must be patent to any person who has suffered from a bad toothache, during which if sensations could be trusted the tooth becomes of enormous size, to diminish gradually as the pain subsides. Nothing is so deceptive in a general way in matters of diagnosis as the impressions of a patient with regard to the progress of his disease—although sometimes they are remarkably correct—and you must exercise great caution in estimating their value, or you will be constantly in danger of making grave mistakes.

June 11th, 1906.

ALL other attempts to relieve a patient during a bad attack of hay fever having failed, Wolner applied cold compresses to the man's forehead and face, wringing them out of ice water, and reapplying them as soon as they began to get the least bit warm. In three-quarters of an hour relief was obtained. The treatment was kept up constantly for about three hours, and off and on for about six hours. A year later the attack came on again. The patient applied cold compresses and was relieved.—*Journ. A. M. A.*, vol. xlvi, No. 21.

A LECTURE

ON

SOME ACID INTOXICATIONS.

Delivered at the Medical Graduates' College and Polyclinic.

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GENTLEMEN,—It has been said that any biological problem, if probed deeply enough, becomes a chemical one. Without fully accepting this statement, we may agree that the importance of the chemical factor in disease is becoming increasingly recognised. For example, the essentially chemical causation of multiple peripheral neuritis is generally accepted.

Of late much attention has been paid to chemical bodies produced within the organism, which are believed to cause auto-intoxication. Among these are found certain acids, such as sarcolactic, oxalic, carbolic, and uric acids, but I propose to confine my remarks to the diacetic group, because I think we are beginning to get a grasp of their general significance.

I shall deal with the theoretical aspects as briefly as is compatible with making clear the underlying principle of acid intoxication, then discuss some clinical conditions in which this particular form occurs, the significance which I believe attaches to it, the signs by which it may be recognised, and the treatment by which it should be met.

I must ask you to forgive me if I seem a little dogmatic, but I want to put a definite point of view before you, and in the short time at my disposal we could reach no conclusion if we considered all other possible explanations of the phenomena I shall discuss.

An acid intoxication might produce its ill effects either because the acid is a direct poison or simply because it is acid—*i.e.* it upsets the normal alkalinity of the blood. The acids that we are discussing do not fall into the first group; they are not direct poisons, for they can be taken by the mouth in considerable quantities without injurious effect. In the circulation they are harmful simply because they are acid.

Let us see first what happens when acids are introduced into the circulation of animals. If an

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acid is injected into a carnivorous animal the tissues defend themselves against the acid by producing ammonia to neutralise it, and the ammonia salts are increased in the urine at the expense of the urea. In an herbivorous animal this protective ammoniacal formation is less marked. The excess of alkaline carbonates in their blood protects them to some extent against acid intoxication, but, when these have been used up, fresh doses of acid cause a lowering of the alkalinity of the blood. Its power of taking up CO_2 is thereby seriously diminished, for the fixed acid is combined with the bases in the plasma. Therefore CO_2 is retained in the tissues and internal asphyxiation is produced, respiration being first quickened, then slowed. Convulsions may occur and coma results.

From these experiments we may conclude that an early sign of undue acid-formation in the body will be an increase in the amount of nitrogen in the form of ammonia, and a decrease of nitrogen in the form of urea in the urine, the ammonia being combined with the abnormal acids. Later we should expect diminished alkalinity of the blood, a diminished output of CO_2, accompanied by persistence of the abnormal acids in the urine, and the onset of coma. The degree of tolerance to an acid intoxication will depend on the power of protective ammoniacal formation. Fortunately, in man this power is very considerable.

Acidosis in diabetes.—I will take this first because it was in this disease that the condition of acidosis was first recognised, and because it permits us to draw certain general conclusions which we can test in the light of other clinical conditions in which acid intoxication occurs.

That acetone and diacetic acid appear in diabetic urine when the case is not going on satisfactorily is well known. Generally speaking, a rigid dietary increases the amount of these bodies, while the addition of carbohydrates to the diet, though it may increase the sugar in the urine, will usually diminish these abnormal bodies. If more than half a gramme of acetone is excreted in the twenty-four hours, we are certain to find diacetic acid present. If much diacetic acid is present, β-oxybutyric acid will be found also. In severe cases, 20, 30, or more grammes may be excreted in the day. The sequence of events seems to be as follows. The oxybutyric acid breaks down into diacetic acid; the diacetic acid breaks down into acetone. If there is only a small quantity of abnormal acid being formed, the whole will pass down to a state of acetone before it is excreted, and only acetone will be found in the urine. On the other hand, if there is more than a small amount of acetone some of it will not have broken down but will be excreted as diacetic acid. With an increased amount of acetone there will be oxybutyric acid present as well. The chemical changes involved in these decompositions are not very great. By taking away H_2 from oxybutyric acid we get diacetic acid, by taking away CO_2 from diacetic acid we get acetone.

There is no simple test for oxybutyric acid, but we may regard it as probably present if there is a well-marked diacetic acid reaction. On the other hand, when acetone alone is present the acid intoxication can be but slight. We may be quite sure that if any considerable quantity of abnormal acid is being formed diacetic acid will be present. That is fortunate, because it is the easiest of these substances to test for. I want to call attention to one or two points in connection with the testing of urine for these bodies. I have here urine from a diabetic patient, and you will see that on the addition of a solution of perchloride of iron a very distinct red reaction takes place in it. It is generally stated that you can take the colour up with ether, thus distinguishing it from other substances which might lead to confusion. But, as you will see, the ether does not take it up readily.

I need not remind you that if the patient is taking salicylate of soda this reaction will be obscured completely by the violet colour which the salicylate of soda gives to the urine. Another point about this reaction is, that if you boil the urine for a minute or more first, you will not get the colour because diacetic acid will be volatilised. This is important, because the things for which you might mistake diacetic acid will not be affected by boiling. With these precautions I do not think there are any fallacies in this perchloride reaction and, therefore, it is one which is very simple and reliable.

With regard to acetone, perhaps you will think that in view of what I have said the testing for acetone is a matter of no great importance. Well, when an acid intoxication is disappearing it is rather important to know whether there is still acetone present. Let me show you the acetone reaction.

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To the urine I add a few drops of a saturated solution of nitro-prusside of soda and then caustic soda, and a bright-red colour is developed. I now add some acetic acid to it and the colour is deepened and tends to purple. A substance which will give a somewhat similar reaction is creatinin, but the creatinin colour is destroyed by the acetic acid, whereas the acetone colour is increased by it. Acetone is a very volatile substance and, therefore, can be removed very largely by boiling the urine. Herein again it differs from creatinin.

With regard to the significance of these bodies in diabetes, we may say that if a patient is put suddenly on a very restricted diet these bodies are almost certain to rapidly appear, and moreover the patient may begin to show some signs of coma. The symptoms summarised are, that the sudden withdrawal of carbohydrates from the diet of a diabetic patient is followed by nausea, vomiting, loss of weight, and the odour of acetone in the breath, the presence of these acids in the urine, and a great increase in the amount of ammonia in the urine. Though the total amount of nitrogen is not altered, the amount of ammonia is greatly increased. In one case * it is recorded that the ammonia increased from 7 to 29 per cent., and the blood also became less alkaline and contained less carbon dioxide. Then the patient was given more carbohydrates, and these symptoms disappeared. A great improvement took place in the general condition of the patient, including the disappearance of the smell of acetone in the breath and the diminution of the ferric chloride reaction of the urine, although there was no marked change in the amount of sugar excreted in the urine.

Now, what is the source of these bodies in a case of diabetic coma? They do not come from carbohydrates, because carbohydrates diminish the amount of them in the urine. Do they come from proteid or from fat? Both these views have been held. It seems clear that the increase in the amount of nitrogen in the urine is insufficient for them to have come from proteid alone. The view that they come from fat has, perhaps, more to recommend it. von Noorden † takes the following view. Normally, the fragments of the proteid, and the fat molecules that contain few carbon atoms, should be oxidised down to CO_2, but they are only thus oxidised if there is plenty of oxygen present. We know that carbohydrate contains for its weight a larger amount of oxygen than other food-stuffs. He regards the oxygen contained in carbohydrate as an important adjunct to the proper oxidation of the other food-stuffs. According to this view, diacetic acid means deficient oxidation of the tissues. There are certain other facts which point in the same direction. For instance, in mountain sickness people pass diacetic acid in the urine. In such cases the symptoms are associated with diminished oxygen tension in the atmosphere.

The difficulty is that the worst cases of diabetes cannot utilise either the sugar of the food or the sugar produced by their own tissues. It is no use, of course, pouring in the carbohydrate when it is not metabolised. But is the loss of the power to metabolise carbohydrates absolute in diabetes? This can be determined by cautiously giving carbohydrate and ascertaining whether it increases the amount of sugar in the urine, while it diminishes the amount of acetone and diacetic acid.

Other observers refer the acetone bodies mainly to the breaking down of fat, and they find that an increase of fat in the food means an increase of acetone produced in the urine, and, on the other hand, a diminution of fat in the food leads to a diminution of acetone in the urine. I think, therefore, it is advisable to diminish the amount of fat in the diet under these conditions, and to cautiously increase the amount of carbohydrate.

What form of carbohydrates should you employ in the acidosis of diabetes? I mention two which can be metabolised when other forms can not. One is levulose, which certain patients can metabolise rather than dextrose. von Noorden strongly upholds oatmeal as the carbohydrate to be used. He says that oatmeal is tolerated when other forms of carbohydrates are not. It is very much easier to give patients oatmeal than levulose, and furthermore, oatmeal is much less expensive. The first indication in treatment, then, is to limit the production of acetone bodies by favouring oxidation in this way, the second to neutralise the acids already formed by giving alkali. von Noorden gives considerable doses. He recommends half an ounce of bicarbonate of soda in the day, and forty-

* Beddard and Spriggs, 'Proceedings of British Medical Association,' July 27th, 1904.

† von Noorden and Mohr, 'Disorders of Metabolism and Nutrition,' Part IV—"Acid Autointoxications."

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five grains of calcium carbonate as well, because calcium salts are diminished in acidosis. I saw a case only this morning that is having one ounce of sodium bicarbonate a day for this condition. I usually give citrate of potash as well as bicarbonate of soda, because it is not neutralised by the gastric juice, and it becomes bicarbonate in the blood, which is where the alkali is most needed. It can be given in 45 gr. doses. Treatment should be continued until the urine is distinctly alkaline. If, in spite of this, coma comes on, an infusion of sodium bicarbonate of 1 or 2 per cent. may be safely employed. It should not be given subcutaneously but intravenously, and at least a pint should be injected. The total amount of alkali should not be more than about one ounce a day. Beyond this amount it may have a depressant effect on the heart.

Acidosis in recurrent vomiting.—In 1882 Dr. Gee called attention to cases of fitful and recurrent vomiting of unknown causation in children. Later observers noted acetone in the breath, urine, and vomit. Diacetic acid and oxybutyric acid have also been found when looked for. The analysis of fifty-five cases by Batty Shaw and Tribe* gives the following clinical features. The cases usually occur between three and eleven years of age. The frequency of the attacks is very variable, a common interval being three months. There may be a prodromal period in which dyspnœa, sighing respirations, offensive breath, choreic movements, and general restlessness have been noted. The tongue may be either coated or clean. Then vomiting begins, without nausea, and usually without gastric pain, all food is rejected, and towards the end of the attack bile appears, sometimes even blood. Constipation is common. The attacks may last only a few hours, but the average duration is five or six days. It is the rule for fever to occur during the attacks of vomiting. Wasting is often a very marked symptom. In later life these attacks are sometimes replaced by migraine, but they tend to disappear when puberty is reached. Three of the fifty-five cases were fatal. In 1901 Marfan suggested that there was some nutritional disturbance which is either the cause or the effect of an intoxication. Edsall two years later referred the symptoms to acid intoxication, with consequent diminished alkalinity of the blood.

* Shaw and Tribe, 'Brit. Med. Journ.,' February 18th, 1905.

Reviewing the whole evidence, it appears most likely to me that the primary event is a disturbed metabolism so that the tissues are practically starved for the time being. This is shown by the rapid wasting that occurs. We have seen that the abnormal breaking down of the tissues which follows disturbance of metabolism results in the formation of these abnormal acids. Once they are formed the ordinary events of acid intoxication follow.

I do not say that the tissue starvation is simply the result of persistent vomiting, though this no doubt intensifies it; the starvation occurs from the perverted metabolism, which affects all the cells of the body. As yet we cannot tell why this perversion of metabolism occurs, but from the resemblance of these cases in several particulars to the pernicious vomiting of pregnancy which I shall discuss next hepatic insufficiency will, I think, ultimately be found responsible. The toxic element in the case is further suggested by the occurrence of albuminuria and the presence of casts in the urine.

The following treatment is recommended by Gee, Shaw, and Tribe: To ward off attacks, prodromal signs should be noted—white stools, offensive breath, some change in the complexion, usually indicative of what are called "bilious attacks," and the presence of abnormal acids in the urine—then mild aperients should be given and easily digestible food. In most attacks barley-water and veal and chicken broth are best tolerated. Small doses of grey powder or calomel should be given, also bicarbonate of soda, up to three drachms in the day. Normal salt solution *per rectum* is useful. The only criticism that I should venture to make is that the chicken broth would be rich in extractives, and moreover I should think arrowroot might be added, on the principle that the abnormal acids are checked in their production by the administration of carbohydrates.

Acidosis in the pernicious vomiting of pregnancy.—It is now recognised that though the pernicious vomiting of pregnancy may be due in some cases to mechanical causes, such as displacement of the uterus, and in others to neurotic causes, there remains a more serious group in which a toxæmia is responsible. I would urge that it is never safe to refer the pernicious vomiting to mechanical or neurotic causes unless toxæmia can be definitely excluded. In these toxæmic cases necrosis and de-

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generation of the central portion of the liver lobule and necrosis of the excretory portions of the kidney have been found. Whitridge Williams* found also a striking increase in the percentage of nitrogen eliminated as ammonia which, compared with the total nitrogen of the urine, amounted to 16 to 32 or 46 per cent. instead of the normal 3 to 5 per cent. The total output of nitrogen remained about the same in these cases. He suggested that this might be due to either failure of urea-formation in the liver or to the attempt to neutralise acid intoxication. Observations by others show that this latter view is probably the correct one.

Dr Helen Baldwin † found that in such a case the urine yielded a marked reaction for acetone and diacetic acid, but no sugar. The patient's condition was so serious that labour was induced. After this the abnormal acids diminished until only acetone was found. On the tenth day after the induction of labour there was a return of severe headache, nausea, and vomiting, and it was noteworthy that on this day diacetic acid was again found in the urine. After this recovery was uninterrupted.

Dr. Longridge ‡ has noticed a diminution of alkalinity of the blood in eclampsia, but he did not state whether this was due to the presence of diacetic acid. He gave citrates with the object of bringing up the diminished alkalinity of the blood to normal. As I have already said, I have found this method efficacious in the acid intoxication of diabetes. Another point of interest was that sugar was given by the mouth and rectum "in order to replace the glycogen in the liver without which that organ could not exert its antitoxic function." I do not know what evidence we have that the liver cannot exert its antitoxic effect without glycogen. On the other hand, we know that carbohydrates have a distinct influence in preventing the production of these abnormal acids, and I would suggest that the sugar was effective for this reason.

Gastro-intestinal acidosis.—I should just like to mention one case which I saw last year, that of a married lady æt. 24 years, who had been wasting rapidly, with marked constipation and occasional vomiting. When I first saw her she weighed only 5 st. 6 lb. On physical examination she showed the following points. The tongue was very raw; at the right apex the note was impaired and the breath sounds were harsh and prolonged, but there was nothing pointing to active mischief. She had not had any cough for a long time. She had been seen by Dr. Wethered, who also had come to the conclusion that the lung was not responsible in any way for the symptoms. There was also a question as to whether she was pregnant. Two weeks later she had lost two pounds more and there was no doubt that she was pregnant. Diacetic acid was found in the urine, but no sugar or albumen. She was still vomiting, but in consultation with Dr. H. Williamson we found no evidence that it was the pernicious vomiting of pregnancy. Then membranous casts were found in the stools. I ascertained that she had been taking an extraordinary diet—enormous quantities of beef-tea and extractives of all sorts. When she came back from a drive they gave her such things as mushrooms on toast. There was very little nourishment in the diet and an enormous amount of stimulating substances. The treatment was simply to give her a plain but liberal diet, with alkalies before meals and twenty grains of citrate of potassium three times a day. The diacetic acid rapidly disappeared and the patient gained weight—one stone in six weeks—and the pregnancy was going on quite normally. In this case we cannot refer the whole condition of the patient to pregnancy because the vomiting had occurred before pregnancy and even before marriage. My view is that membranous colitis had caused both the emaciation and the acid intoxication, and the condition had been accentuated by faulty diet. I believe the case to have been one of gastro-intestinal acetonuria.

Acid intoxication may be met with in several other conditions, which time will only allow me to mention briefly. First, you may meet with *acetonuria in febrile conditions*. In two cases of typhoid fever von Noorden gave patient A a diet with very little carbohydrate in it and patient B one with plenty of carbohydrate. A had a marked diacetic reaction and B only just a trace of acetone. After a few days the diet was reversed, with the result that the former showed only a trace of acetone and the other a well-marked diacetic reaction. There is little doubt that here, again, the acetonuria was

* Whitridge Williams, 'Lancet,' 1905, vol. ii, p. 1172.

† See 'Lancet,' 1905, vol. ii, p. 1491.

‡ *Ibid.*, 1905, vol. ii, p. 1405.

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due to deficient nutrition, particularly when associated with deficiency in the carbohydrate diet.

Another condition in which we find acidosis is that of carcinoma of the digestive organs or elsewhere. There, again, the loss of nutrition is self-evident. We also find acidosis in uræmia, chronic lead-poisoning, in prolonged rectal feeding, and in some psychoses. In all these the element of malnutrition is obvious.

There is one more condition of special interest, namely *acid intoxication after anæsthetics*. Dr. Leonard Guthrie,* in 1893, called attention to the condition of delayed chloroform poisoning. During the last eighteen months much more attention has been paid to it, and it has been shown to occur sometimes after ether as well as after chloroform. The description which he gives of the condition is this: Twelve hours or so after the anæsthetic the patient (usually a child) suffers from profuse and repeated vomiting, the vomit eventually resembling the dregs of beef-tea, restlessness, excitement, delirium, alternating with periods of apathy, jaundice (occasionally), and unconsciousness, deepening to coma. Death usually occurs about the fifth day, but sometimes later, from cardiac or respiratory failure, gradual or sudden. Pyrexia is not marked as a rule. Albuminuria and the presence of casts in the urine are common. Furthermore, there is a smell of acetone in the breath, while acetone and diacetic acid are found in the urine. These substances are only looked for, as a rule, in cases, after chloroform, where something goes wrong. In order to test whether they appear without such symptoms, Mr. W. G. Ball, house-surgeon at the Metropolitan Hospital, kindly examined for me the urine passed before and after an anæsthetic in all the operation cases during three months. Not a single case showed diacetic acid, so that I think we may conclude that its presence is significant. Hubbard,† however, found acetone nine times, and in four with diacetic acid, out of 135 surgical cases. In another series of seven cases acetone or diacetic acid were found, on being looked for, to explain some irregularity in convalescence. Becker found acetone after anæsthesia in healthy persons. We need, therefore, only attach importance, I think, to the presence of diacetic acid. The nature of the complaint which necessitated operation seems to have little or nothing to do with it. Fatty degeneration of the liver, muscles, and kidneys, but especially in the liver, has been found post mortem in these cases.

What is the cause of this fatty change? Guthrie holds that it must have existed prior to the anæsthetic, which merely acted as the "last straw." If this be so, it is possible to understand why anæsthetics should be dangerous at one time and not at another, the element of danger being the superabundance of fat existing at the time of operation. He further suggests that the storage of superfluous fat in the liver may be due in some cases to the large quantity of cod-liver oil and fattening diet supplied to delicate crippled and rickety children in the hope of strengthening them, coupled with want of exercise. It is interesting to remember that both ether and chloroform are solvents of fat. Some other people have held the view that the condition is primary and wholly due to the chloroform (Stiles and McDonald, Carmichael, and Beattie).

The advantage of Guthrie's view, that the chloroform merely acts as the "last straw," is that it explains the condition without any reference to idiosyncrasy. Idiosyncrasy will hardly explain why a patient takes chloroform well the first time and shows bad symptoms when chloroform is taken a second time in much smaller doses. I think the most satisfactory view to take is that there must have been some fatty degeneration of the liver first. In one case which was recorded the liver could be felt three fingers' breadth below the costal margin twenty-four hours after the administration of a small dose of chloroform. This could hardly have been the direct result of the chloroform.

The suggestion in these cases is that the first administration of the anæsthetic caused the fatty degeneration, while the second administration, which in many cases had only been of a very small quantity, had served to dissolve the fat and throw it into the circulation. Droplets of fat have been actually seen in the alveolar septa of the lungs.

Guthrie suggests as a line of treatment (1) that before operation on even fat and apparently healthy children careful inquiry should be made as to the history of so-called "bilious attacks," which may in reality be those of acidosis. (2) In all cases

* Guthrie, 'Lancet,' 1894, vol. i, pp. 193 and 257; 1903, vol. ii, p. 10; 1905, vol. ii, p. 583.

† Hubbard, 'Boston Medical and Surgical Journal,' June 29th, 1905.

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where over-fattening and want of exercise are suspected operation should be delayed if possible till the patient has been subjected for some days to a fat-free diet. The urine should be examined for diacetic acid, and if present a course of alkalies should be prescribed. (3) Both starvation and fright cause acetonuria. He thinks that the four hours' fast for children before operation is too long. He recommends the giving of nutrient enemata after the lower bowel has been cleared, two hours before and immediately after operation. The effect of fright cannot be altogether controlled but may be combated by preventing starvation. Should symptoms of acid intoxication occur despite these precautions, it must be treated as in other cases.

Now as to the *conclusions* arrived at.

(1) Diacetic acid appears in the urine under clinical conditions which, though differing widely, have these points in common:

(*a*) Relative starvation of the tissues; broadly, that they are being starved of carbohydrates or, as in diabetes, are not able to use the carbohydrates presented to them.

(*b*) Excessive disintegration of the fatty tissues in the body. Really these two conditions are often the same, for if the tissues are being starved the body attempts to live on its own fat. The *rôle* of the carbohydrate, in von Noorden's opinion, is to provide the tissues with oxygen which it contains in relative abundance. In this connection I may remind you of the appearance of abnormal acids in a condition of simple oxygen starvation—*i. e.* mountain sickness. Our first duty in acid intoxication, then, is to find out why the tissues are being starved and wherein the diet is at fault.

(2) The simplest test, and the one that gives us the most information, is the claret colour given by the urine on the addition of perchloride of iron.

(3) The further production of these bodies should be prevented as far as possible by cutting off fat in diet and by adding carbohydrate. In diabetes levulose (fruit sugar) may be utilised up to 50 or 100 grammes a day, but according to von Noorden oatmeal gruel is as efficacious, and it is much cheaper and more easily obtained.

(4) The toxic effects of these acids should be neutralised by administering bicarbonates freely, if necessary up to one ounce a day. Personally, I like to give some of the alkali in the form of citrates which may be given in 45 grain doses. If coma supervenes, venesection should be performed, followed by intravenous, not subcutaneous, infusions of at least a pint of 1 to 2 per cent. of bicarbonate of soda.

I would ask you to test these conclusions as opportunities may arise, for I believe that in acid intoxications we have the explanation of symptoms that would in many cases otherwise remain obscure.

June 11th, 1906.

PRURITUS: THE DIAGNOSIS OF ITS CAUSES, AND ITS TREATMENT.

By H. G. ADAMSON, M.D.Lond, M.R.C.P.,
Physician to the Skin Departments at Paddington Green Children's Hospital and at the North-Eastern Hospital for Children, and Assistant in the Skin Department, Westminster Hospital.

ITCHING is a symptom which may occur not only in many diseases of the skin, but also in connection with many constitutional and visceral diseases. In certain skin affections it is often the most prominent feature, the main reason the patient has for seeking advice, and its cure the chief aim of our therapeutic efforts. In other instances it may furnish a clue to the discovery of some important general or visceral disease, and this fact should never be lost sight of in the presence of a case of pruritus.

From an etiological point of view cases of pruritus may be divided into four groups:

(1) Those in which the itching results from the presence of animal parasites.

(2) Those in which the itching is a symptom of some definite skin affection.

(3) Where it results from abnormal constitutional conditions of diseases of internal organs.

(4) Cases in which no local source of irritation can be discovered, in which there is no skin affection and no evident disease of internal organs, such cases as have been called "mental pruritus" or "neurotic pruritus."

In the large majority of cases pruritus, when not due to animal parasites or to an obvious skin affection, is a symptom of some internal disease or functional disturbance, and every effort should be made to discover this, since the ultimate cure of the itching usually depends upon the removal of the cause. No case should be regarded as neurotic or mental until every other source has been excluded.

For clinical convenience pruritus is usually divided into two classes, viz general pruritus and local pruritus.

(I) Strictly speaking, the term "general pruritus" (*pruritus universalis*) is reserved for those cases of more or less generalised itching which do not depend upon local irritants or upon some form of

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skin disease. Practically it is better not to make this distinction, but to regard the terms "pruritus" and "itching" as synonymous.

In investigating a case of itching or pruritus with the view to making a diagnosis of the affection which leads to it one must first of all exclude all local parasites, especially those of *pediculosis* and of *scabies*. These are amongst the commonest causes of pruritus, and no station in life protects from them. Scabies may occur at any age. A very common error is to mistake scabies for eczema, chiefly because the patient's position in life seems to exclude it. In all cases of generalised itching this complaint should be first thought of, and the wrists and hands should be examined for scratch marks or for the burrows of the acarus. In males the burrows may often be found upon the penis and in children upon the feet. *Pediculosis* is seen almost exclusively in elderly people, and especially in elderly alcoholics. Scratch marks about the shoulders should be looked for and the punctate hæmorrhages produced by the mandibles of the louse. It may be difficult to discover the insect in the folds of the linen.

If a *skin eruption* is present one must be careful to remember that this may be the result of the scratching; for example, impetiginous or eczematous lesions may result from the scratching in scabies. In long-continued pruritus the skin may show papules, erythematous patches, and scratch marks, and, after a time, more or less pigmentation. Of the *skin eruptions* in which pruritus is a prominent symptom the chief are, eczema, Lichen planus, and urticaria, and the less common but serious affections, Dermatitis herpetiformis and the pre-mycosic stage of Mycosis fungoides; in the presence of a long-continued dry eczematous condition accompanied by pruritus it is important to bear in mind the question of Mycosis fungoides.

Having satisfied one's self that the itching is not the result of animal parasites, and when it is not due to a definite skin disease, investigations should then be made into the general condition of health and the state of the viscera.

In aged people one meets with what is called *senile pruritus*. It seldom occurs before the age of sixty-five years, and it is usually associated with senile atrophy of the skin, though chronic alcoholism and pediculosis are often found as exciting causes.

In middle-aged persons, especially between the ages of forty and fifty, pruritus is often associated with the *lithæmic or gouty condition* or in many cases with symptoms of *chronic dyspepsia* and *constipation*.

In female patients it may accompany some *disturbance of the sexual organs*. It is especially common at the *climacteric*, and it may occur in connection with *pregnancy*.

It is of the utmost importance to remember that pruritus may also depend upon more *serious affections of the internal organs*, such as malignant disease of the liver, of the gall-bladder, of the stomach, or of the uterus. It not seldom occurs as a symptom of *diabetes* or of *Bright's disease*, and in every case of persistent pruritus the urine must be examined for the presence of sugar or albumen. It is well known as a frequent accompaniment of *jaundice*. Again, itching may be a symptom of *tuberculosis* or of *heart disease*.

In the case of a prolonged and obstinate pruritus even when no possible cause has been discovered the prognosis should be guarded, since in many of these incurable cases of pruritus occurring in middle life there eventually develops malignant disease of some internal organ.

There is a not very uncommon form of pruritus known as wintry pruritus (*pruritus hiemalis*) which occurs in some persons during the winter months. It may be general, but it affects particularly the external surfaces of the limbs, and especially the thighs, and it is generally worse on exposing the body to cold—that is, on undressing or getting out of bed. People who suffer from this form of pruritus are generally neurotic or gouty.

(II) The second clinical group of cases includes all local forms of pruritus. *Local pruritus* occurs usually at one of the natural orifices, or rather at the junction of skin and mucous membrane. Itching at the margins of the eyelids, at the urethral orifice, and at the nose are all examples of local pruritus. The most important of these, however, are *pruritus ani* and *pruritus vulvæ*.

Pruritus ani occurs usually in middle life or at a more advanced age. Its commonest causes are eczema, local congestion from piles, or fissure of the anus. In many cases there is also some digestive disturbance of an hepatic or gouty nature, and sometimes this may act alone without any local trouble. Occasionally it is excited by some article

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of diet, such as alcohol in the form of spirits, coffee, or red pepper.

Pruritus vulvæ may also depend upon a local cause, as eczema, vaginitis, thread-worms, or irritating discharges from the cervix or uterus. It may arise also as a reflex from uterine or ovarian disease and it may be due to pregnancy of the uterus. The local irritation of diabetic urine is a cause which must not be forgotten, and one should never omit to examine the urine for sugar. Sometimes careful search fails to find any local or reflex cause, and such cases are very obstinate to deal with.

To sum up, in investigating a case of pruritus, unaccompanying an obvious skin eruption we should (1) first of all exclude scabies and pediculosis; (2) search for evidence of disturbance of the digestive functions; (3) examine the urine for sugar and for albumen; (4) examine the chest, heart, and abdomen for evidence of organic visceral disease; (5) when necessary examine the uterus and ovaries in a female; (6) search carefully for any local trouble in pruritus ani and pruritus vulvæ.

Treatment.—The diagnosis of the cause of the pruritus is the first step towards its successful treatment. When the cause cannot be discovered and removed, the prognosis is bad. In all those cases in which the pruritus is associated with a disease of the skin an important part of the treatment is to prevent the irritation of the skin by the scratching which the pruritus induces, since such irritation generally tends to aggravate the disease and to prevent its cure. Hence the great value of protective applications in eczema and of anti-pruritic applications in urticaria and in Lichen planus or prurigo. Some writers advise the administration of narcotics, and in cases of infantile eczema chloral may be given at bed-time in doses of fifteen to twenty mins. of the syrup to a child of six months, or half a drachm to a child of one to two years. In older persons bromides are better and safer than chloral. Opium should seldom be given, as it usually tends to aggravate the itching. Such drugs are, however, seldom required if proper precautions be taken to protect the eczematous areas from irritation by exposure, by clothing, and by scratching.

In pruritus due to *scabies* appropriate treatment for the affection usually quickly gets rid of the itching, or if it persists after the cure of the scabies a mild tar lotion is effectual in removing it.

Senile pruritus is sometimes relieved or even cured by the avoidance of alcohol or by the removal of pediculi, and an excellent and cleanly method of treating pediculosis of the body is that of making the patient wear a peice of sulphur in a small muslin bag slung from the neck. Those cases of senile pruritus to which no cause can be assigned beyond the atrophy of the skin are of much less favourable prognosis; many of them can be relieved by an empirical remedy, viz. cannabis indica, beginning with five minims of the tincture three times a day and increasing to twenty or thirty minims for each dose. It must be well diluted and taken after meals.

In pruritus dependent upon disturbance of the digestive functions the bowels must be regulated, and careful attention given to the diet, alcohol being avoided and any particular kind of food which has been found to disagree.

In pruritus of diabetes, of albuminuria, and of cardiac disease, these diseases must, of course, receive the first attention. Cardiac cases are often relieved by small doses of digitalis. Winter pruritus will often cease on stopping the cold morning bath and adopting a warmer form of clothing. In many cases of pruritus cod-liver oil internally is useful as improving the general condition of the skin.

As empirical remedies in obstinate cases, or in cases where the trouble is due to organic disease, or as temporary measures in all cases one of the following drugs may be chosen: quinine in large doses, antipyrin or phenacetin in doses of gr. x and gradually increased.

In intractable forms of pruritus injections of pilocarpin are often of great service. In senile pruritus especially the injections may be continued for weeks or months, and then seem to do permanent good, possibly by increasing the nutrition of the skin by constant dilatation of the capillaries. The immediate effect in allaying irritation is probably due to the sweating induced, which relieves the dryness of the skin. The most satisfactory method of employing this drug is as follows: the patient takes a warm bath in the evening just before going to bed, a hot drink is now given and an injection of pilocarpin gr. $\frac{1}{10} - \frac{1}{5}$; profuse sweating occurs and the patient gets several hours sleep, just at the time when the itching is most troublesome. This proceeding may be repeated

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nightly for weeks and sometimes leads to the permanent cure of the pruritus.

In all cases local applications are useful, and the list of such applications is almost endless. Among the most useful are those of the disinfecting type, liq. carbonis detergens, carbolic acid, Sanitas, etc.—of course suitably diluted. To the lotion may be added such powders as zinc oxide or calamine. In many cases cold applications are found to relieve, such as cold sponging with vinegar and water, or with alcohol and ether, and afterwards powdering with starch powder. Lotions may be conveniently applied by means of a spray. A lotion of perchloride of mercury, gr. $\frac{1}{2}$ – 3 to the ounce, is cleanly and odourless. Menthol, gr. 5 – 10 to dilute alcohol ʒj, is a useful lotion.

In *pruritus ani* and in *pruritus vulvæ* the rational treatment is to remove the cause if possible. Fissure of the anus requires surgical treatment. The digestive organs are often at fault in pruritus ani and treatment must be given in this direction. As temporary measures many local applications are of value. Calomel ʒj to ʒj vaseline will sometimes give immediate relief in pruritus ani. It is important to insist upon local cleanliness. In pruritus vulvæ relief may often be obtained by bathing the parts with a warm, freshly-made, saturated solution of boracic acid and, after drying thoroughly, the free application of a simple zinc ointment, though of course here again local or reflex causes should be sought for and relieved if possible. We have now, too, in the X rays a valuable remedy for obstinate cases of pruritus ani and pruritus vulvæ. The application should be made according to the modern methods of dosage; a dose of 3 H to 4 H being given once in ten to fourteen days. Most cases are immediately relieved, and many remain permanently cured.

June 11th, 1906.

CLINICAL OBSTETRICS. By ROBERT JARDINE, M.D.Edin., Professor of Midwifery in St. Mungo's College, Glasgow, etc., etc. With ninety-six illustrations and a coloured plate. Second edition, pp. 609. London: Rebman, Ltd., 1905.

The coloured plate represents a liver from a case of eclampsia. This fact, that the most conspicuous illustration should be of a detail in the morbid anatomy of a somewhat rare case, is characteristic of the work. It is emphatically a book of personal experience and opinion. The author, as is known, has taken a special interest in puerperal eclampsia, a disease in which, many are now coming to think, the changes in the liver are as important as those in the kidney. He advocates the treatment of the disease by the injection into the cellular tissue of saline solution. He relates thirty cases treated in this way, of which four died. If the mortality had been the average, there would have been six deaths. Dr. Jardine, however, claims to have reduced the mortality in Glasgow by one half. If this is so, the mortality in the past must have been exceptionally high. Dr. Jardine teaches that it is necessary, or at least advantageous, to empty the uterus. He brings forward no evidence of this, and there is a large mass of evidence that emptying the uterus in eclampsia does not do good. Dr. Jardine has not apparently yet injected very large quantities of saline solution; two or three pints appears to be the maximum. Why does he not try the six pints recommended by Wooldridge for exhaustion after sevère hæmorrhage, or the fifteen or twenty pints injected by Barnard for septic peritonitis? Turning to another matter, we observe that the author cannot make up his mind as to the relative merits of Barnes's and Champetier's dilating bags. He repeatedly tells the student to use Barnes's or Champetier's. We find the obvious reason at page 182. Dr. Jardine has not been well served by his instrument-maker, who has supplied him with a bag not made according to Champetier's directions, under the name of Champetier's bag. Champetier's bag is not made of indiarubber, but of waterproof silk. Dr. Jardine has had a bag made for himself of this material; but he has misgivings about using it, for he thinks it is difficult to make it aseptic. Why? Surely it can be boiled. Like many others trained in the Edinburgh school, Dr. Jardine seems unaware of the clinical fact that in most cases of retroversion with incarceration of the pregnant uterus, if the patient is kept recumbent, with her bladder empty, the uterus will right itself; although he relates one case in which this happened. Nor does he apparently know that if the retroversion is left unreduced, and treatment is limited to relieving retention of urine, when full term is reached, natural delivery will take place; although Merriman described the process many years ago. The book is a very interesting one.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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SOME POINTS IN THE TREATMENT OF CLEFT PALATE.

By W. ARBUTHNOT LANE, M.S.

THERE are certain cases of cleft palate presenting such conditions as require treatment other than that I usually adopt.

I propose to illustrate some of the varieties met with and the manner in which they can be operated on with most advantage. In the majority of cases the methods of flap-formation illustrated by Figs. 1, 2, 3, 4, and 5 are the best. The security

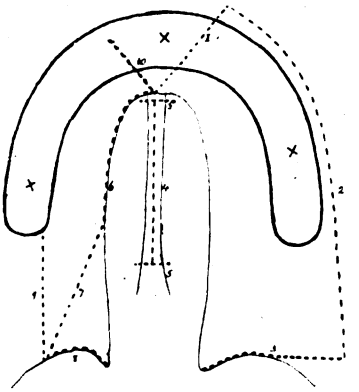

FIG. 1.—Incisions and flaps for broad cleft.

of the flaps depends entirely on the nature of the muco-periosteum forming the margin of the cleft. If, as is usually the case in the young subject, the tissues in this position are thick, the reflected flaps obtain an abundant blood-supply and if skilfully dealt with excellent results are obtained.

It happens not infrequently that it is impossible to close the whole length of the cleft by one single operation.

Fig. 6 illustrates such a condition. The cleft is a very wide one, and it is impossible to close it by the reflection of flaps in the manner described. An incision is made along the entire outer aspect of the gum along the line indicated by 1. Two

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are made along the direction of 2, and two others along the free inner margins of the cleft.

The flap included between 1 and 2 on either side is turned back, great care being taken of its attachment, which is usually very thin. The flaps comprised between 1, 2, and 3 are raised from

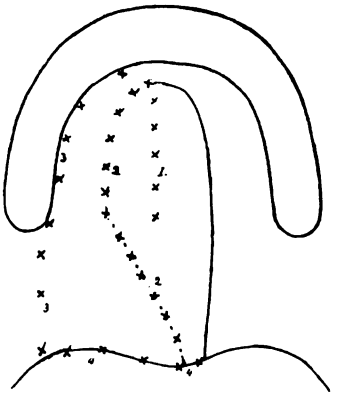

FIG. 2.—Flaps in position.

before backward, care being taken to avoid any damage to the descending palatine vessels. These flaps are then displaced inwards, as in Fig. 7, their apposing margins being sutured together and to the subjacent flap, and if possible to the septum also.

At a later period the posterior portion of the

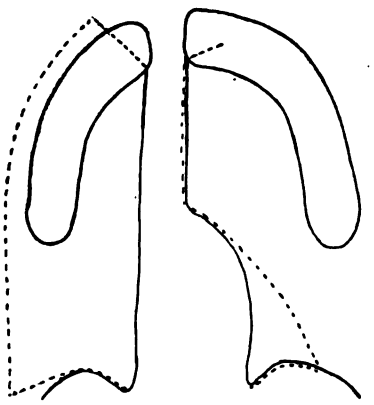

FIG. 3.—Flaps for a common type of cleft palate.

cleft may be closed in one of two ways, the method varying with the breadth of the cleft and the extent of material at disposal. The first and more generally applicable method is by reflecting a flap inwards on one side, leaving it attached by its inner margin, the other flap being rendered raw on its posterior surface and its area extended as in Figs. 4 and 5. The second method is to reflect a flap inwards as before, while the flap from the opposite side is raised from the subjacent parts by an incision extending along its inner, posterior, and outer margins, so that it pivots anteriorly as in Fig. 8, and can be made to cover the flap reflected from the opposite side. Or both flaps may be made to pivot upon their anterior attachments, their

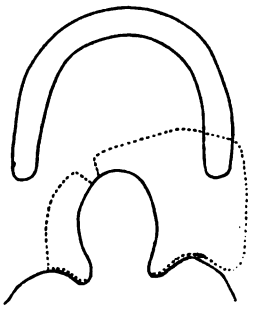

FIG. 4.—The dotted line to the right of the cleft represents the incision along the buccal aspect of the palate, and that to the left the incision along the pharyngeal aspect.

internal edges are sutured carefully together, the outer free margins being anchored wherever a suitable attachment can be found. The free inner margins of the cleft are also pinned down to the superjacent flaps by sutures.

In Fig. 8 I have indicated the mode of closure of the back of the cleft by raised flaps, the outlines of which are represented as dotted lines, and in

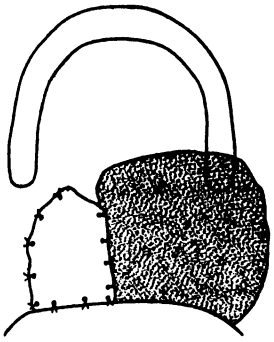

FIG. 5.—The flaps sutured in position.

Fig. 9 the suturing of the raised flaps on both sides in position. In order to avoid confusion, the knots of the sutures which attach the inner margins of the cleft to the superjacent flaps are not indicated in this diagram.

In this diagram the outlines of the flaps are represented as firm lines, the dotted lines representing the position of the incisions, and show portions

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of the area deprived of mucous membrane, and uncovered by the flaps, which have been brought inwards so as to appose their inner margins.

Occasionally in clefts involving the soft palate, with probably a proportion of the hard palate, the soft parts forming the anterior two thirds of the

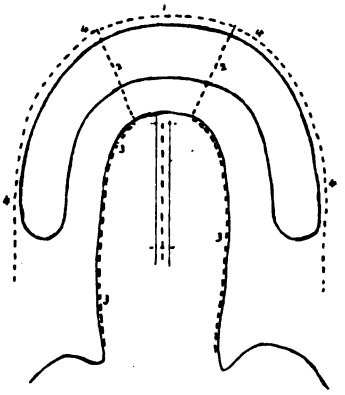

FIG. 6.—Flaps for a very wide cleft.

margin of the cleft are as thin as paper. In such circumstances the splitting of the flaps can only be effected up to a considerable distance from the free margin, or some means other than splitting must be adopted. In splitting a thin flap there is a risk of a failure of nutrition of a part of it so that a hole remains at the anterior limit of the cleft.

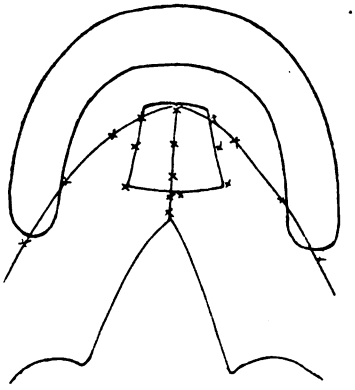

FIG. 7.—Flaps in Fig. 6 in position.

An aperture in this situation is often very difficult to close. I have met this difficulty most effectually by dealing with the cleft in a manner which at first sight appears to be more complicated than it really is. The object is to employ a large flap whose vitality is of a very high order, and this is insured by its containing the descending palatine vessels and nerves of one side.

Fig. 10 shows a cleft involving the soft and part of the hard palate. On the left half of the diagram an incision represented as a dotted line commences well outside, and behind the aperture through which the descending palatine vessels emerge. From that point it is carried

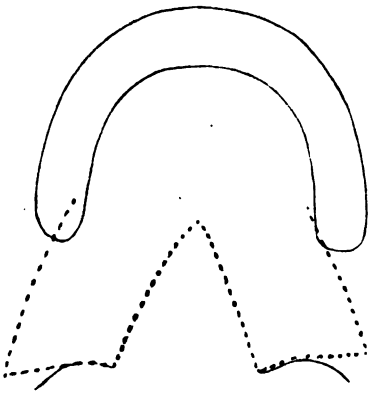

FIG. 8.—Flaps for closure of back of cleft.

forwards along the alveolar margin across the middle line, and along the alveolar margin of the opposite side. It then extends along the outer limit of the soft palate and its lower free margin. Another incision is carried outwards and slightly forwards from the anterior limit of the cleft to meet the first incision, and another

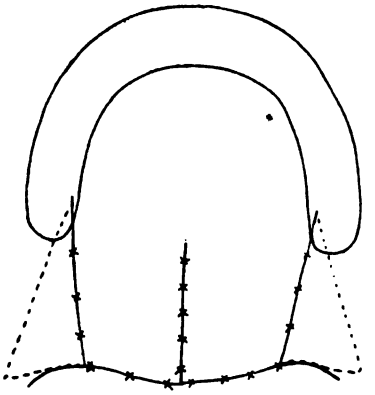

FIG. 9.—Flaps in Fig. 8 in position.

from the tip of the uvula along the free margin of the soft palate to the commencement of the second incision.

The four-sided flap of the soft parts forming the anterior surface of the palate is raised and reflected inwards to the free margin of the cleft. The large flap containing the vessels and nerves

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is raised from the hard palate and is separated from the margin of the cleft in the hard palate. The mucous membrane is reflected outwards from the posterior surface of the soft palate from the margin of the cleft so as to leave a raw area of about a third of an inch broad.

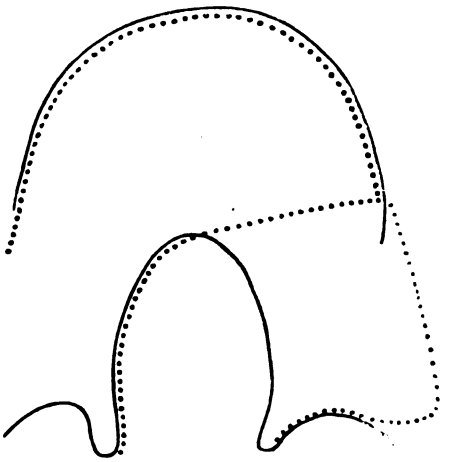

FIG. 10.

The quadrilateral reflected flap of soft palate is pinned by deep sutures to this bare surface, by means of which sufficient areas of the raw surfaces on either side are brought securely into accurate apposition (see Fig. 11).

The large flap is then buckled up so that its

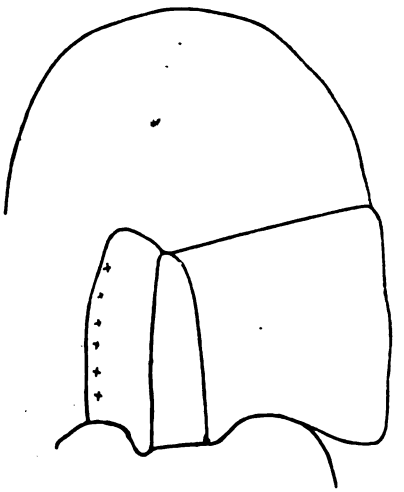

FIG. 11.

lower margin comes into apposition with the free margin of soft plate on the same side. These edges are then retained in apposition by sutures which also perforate the surface of the subjacent reflected quadrilateral flap.

The remaining free edge of the large flap is then completely sutured to the long margin of

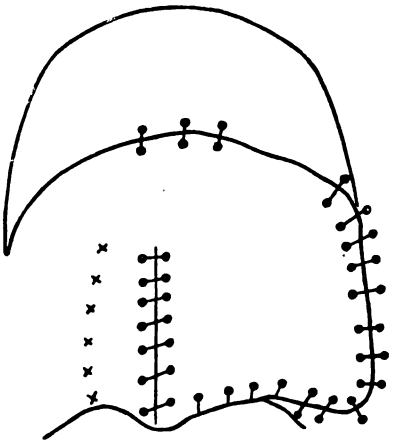

FIG. 12.

the soft parts on the opposite side and to that of the outer limits of the quadrilateral incision (see Fig. 12).

June 18th, 1906.

A LECTURE

ON

LICHEN AND SOME OTHER DISEASES OF THE SKIN.

Delivered at University College Hospital.

By H. RADCLIFFE CROCKER, M.D., F.R.C.P.,
Physician in Charge of the Skin Department.

GENTLEMEN,—The subject of lichen is one about which the mind of the profession is rather in a state of confusion. Many men think that when they have diagnosed a condition and called it "lichen" they have made the diagnosis. As a matter of fact, they have simply given it a name which signifies that it has papules as one of its principal features, and unless they give it a more specific designation, they have not made a diagnosis at all. Another term which is used sometimes is "lichenoid." That is a question-begging term, which really means nothing at all. I propose first of all to sketch briefly the affections to which the term "lichen" has been

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given in the past and those to which it is confined at the present time. In the time of Willan, who was the father of English dermatology, the name was given to anything in which papules were in some degree a feature—a heterogeneous group, now broken up and its members relegated to their proper categories. There was Lichen urticatus, which was really the urticaria of infants, Lichen circumscriptus,, or Lichen circinatus, which is a seborrhœic disease, of which I show you drawings, a very common eruption, always connected with seborrhœa of the scalp, situated between the shoulders and on the chest, which, though papules are a feature in it, is not reckoned in the group lichen now. There was Lichen lividus, which was a form of purpura ; and Lichen tropicus or "prickly heat"; Lichen scrophulosorum, which was the sweat eruption of infants ; Lichen syphiliticus, which especially referred to two forms of follicular syphilis, but the term "lichen" is not now used in connection with syphilitic eruptions. In modern dermatology the group lichen only now comprises half a dozen diseases : Lichen planus, which is the most important of the group ; Lichen acuminatus, a very rare disease ; Lichen variegatus, a still rarer disease ; Lichen scrophulosorum, which, as its name indicates, is specially seen amongst people with a tubercular constitution ; Lichen circumscriptus of Vidal ; and Lichen spinulosus. I do not intend to dwell long upon most of these affections. Two of them, while they are of course interesting and very important to those who are unfortunate enough to have them, are extremely rare, and it is, therefore, very likely you will never see a case as long as you live. I show you pictures of the disease called Lichen acuminatus. There was a great controversy about this for a long time. It was described independently in France and in Germany, though the original case was an English one. The French called it "Pityriasis rubra pilaris" and the Germans "Lichen ruber." Now we know that the two names represent the same disease. It is one of the few diseases which may be universal. There are many diseases which are general—that is to say, spread over every region of the body—but there are very few which occupy every portion of the body. This Lichen acuminatus is one of them. It shows little rounded papules seated in the follicles and more or less closely seated together. These gradually increase in numbers until they coalesce into large areas. On the palms they do not form papules, but diffuse redness, and about the knee-joints they form encrustations resembling very acute psoriasis. Here is one showing the universality of the eruption with rings and papules, following the lines of fission. On the face, you will generally see it with redness and branny desquamation, and about the fingers, especially the hairy parts of the fingers, it forms crateriform papules. It is a rare, obstinate, and troublesome disease, but I have found lately that with thyroid extract great amelioration can be pro duced, combined, of course, with local remedies. That is all I propose to say about it now.

A still rarer disease is this Lichen variegatus, which was first called Parakeratosis variegata. It closely resembles Lichen planus in the matter of individual papules, but it forms a retiform pattern all over the body, and the eruption may occupy every region of the body, though the uncovered portions escape in some patients. It is very persistent, and as far as I know, absolutely incurable. Fortunately, it does not produce any severe symptoms ; it is not associated with great itching usually, and it is not associated with any great sensory disturbance. But sometimes there is itching and burning. The general health, however, is totally unaffected. It occurs in very robust individuals, and very frequently one has been able to trace it in people who are subject to great changes of temperature, such as that of a marine engineer, who was one of my patients. He was subjected to great heat below in the engine-room, and then would come up into the cold air for a time. Several other patients have given a somewhat similar history. I have a patient in private now who has tried the world over to get cured, but I fear he will fail. That, again, is a rarity. Then there is Vidal's lichen, which is not so uncommon. That forms patches chiefly on the nape and sides of the neck and in the groin, but it may be seen elsewhere. The patches are simply broken up into what is called lichenification ; in fact, it is not a true variety because some of these cases go on and develop into a more general Lichen planus ; and others may be traced to chronic eczematous conditions, with thickening of the skin. It is fairly amenable to treatment with glycerine of lead and tar lotions, and similar applications. Local treatment is generally all that is required. Again, here is an illustration of Lichen spinulosus.

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It is an interesting disease which is easily recognised. It forms papules about the hair-follicles, with horny spines one sixteenth of an inch long. They are readily seen by looking sideways, and they can be felt if the hand is passed over them, like a nutmeg-grater. It chiefly occurs amongst children, and its etiology, like that of many other conditions, is obscure. It yields generally to soft-soap treatment combined with emollients applied after the soft soap and spirit have been vigorously rubbed in. It is a disease which gets well slowly, but it always does get well. It occurs chiefly about the neck, the prominences of the shoulders, and the hips. But now and then it is very widespread, as in the young man whose picture I show you, in whom it was all down the thighs and all over the back. Though not very rare, it is certainly not a common disease. Here is a drawing showing the condition called "lichenification." Any chronic inflammation attended with itching and scratching is liable to produce thickening of the skin and exaggeration of the natural lines, with a quadrillation of the lines. It is not a real disease in itself, but is a symptom of chronic inflammation of the skin without visible exudation.

We come now to our *pièce de resistance*. Lichen planus is a very important disease, because, while not one of the most common diseases of the skin, it is yet sufficiently common for every one of you in practice to be sure to meet with cases, and if you are not attentive to what I am telling you to-day, and do not remember it, you will not recognise it and will not succeed in curing it. The most important and typical thing in Lichen planus is the papules of which it is composed. A papule, you know, is a dry elevation of the skin due to inflammation. The great majority of papules are round in contour, and convex or acuminate in projection. But Lichen planus differs from this : it is angular in outline, and it is flat on the surface, hence the name "planus." If you look at your own skin you will see it is broken up by multitudes of small lines into little angular areas. The Lichen planus papule is formed from that ; it is a thickening of the spaces in between these natural lines, due to inflammation, and a gradual elevation above the surface, and a deepening of the natural lines by the thickening of the other portion of the tissue. Several of these may coalesce, or they may simply occupy the actual area of one of these quadrillations, and ultimately a slightly raised flat papule with irregular outline is formed. Sometimes that papule is perfectly square, sometimes it is polygonal, occasionally it may be stellate, having minute processes at the periphery. One of the most typical and at all events suggestive features, besides its angularity and flatness, is the tint of those papules, which develop slowly. In the acute cases these are bright red, but the more slowly growing and more common papules are of a violaceous tint. And whenever you see an eruption of a slightly violet tint you should think of Lichen planus. I do not say it is the only disease which can produce such a tint, but it is the most common and most marked one. Therefore until you are able to exclude that from your diagnosis, you are not justified in going farther. If you would only recollect that one point, that whenever you see a violaceous tint in an inflammatory eruption, Lichen planus is the most likely condition, you would save yourselves many errors. Now, these papules tend to be arranged in various ways. The most common arrangement is in lines, and they are frequently determined by local irritation. The most common local irritation is scratching. If you scratch up and down the arm, these lesions develop in the line of scratching. I have also seen them follow a cat scratch and the scratch of a pin. There are a few rare cases in which the papules have not only been in lines but have been extremely hypertrophic, and formed what has been called "moniliform lichen." But that is a condition which may be regarded merely as a curiosity, as it is extremely rare. Here is an example where it looks like a string of beads along the flexures. This is an extremely exaggerated condition of it. There are not more than a dozen cases on record, and it exhibits the condition which may develop in a person whose skin extremely resents irritation. It is probably more dependent on the idiosyncrasy of the individual than upon the disease itself. Still, there is the fact that it does tend to develop in lines. It may also develop into roundish patches which clear in the centre and form circles, but those circles are generally small, not more than three quarters of an inch as a rule. I shall hope to show you some in patients a little later on. The circular formation, however, is not the most common, but it is curious that those patients who develop these circles in one place are very likely to develop them also in another.

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Therefore in the same patient you will find them multiple, while in the majority of patients the subjects of the disease you do not find the lesions in circles at all. Circles may also be formed by the aggregation of papules into those circles, but in any case they are always small and do not indicate that the case is getting well, as may be said for psoriasis. Psoriasis also clears in the centre, and may spread at the periphery till large circles are formed, but Lichen planus circles never get beyond small dimensions. Another curious thing is that occasionally the disease is distributed in lines following the nerves of the skin. Here is an example extending from the vulva backwards right down the lower limb, very much in a line with the small sciatic nerve. There have been several such cases. I have also occasionally seen it follow the lines of other nerves. Here I show you a picture with it extending along the waist and the arms like a Herpes zoster. Such cases are, however, rare. The positions chosen by Lichen planus lesions are generally the flexures of the wrist, less often the extensor aspect also, over the hips and round the lower part of the body, abdomen, and lower part of the back, the inner side of the knees, especially over the vastus internus, and various parts of the leg. The head generally escapes, and so does the face, but there is no part which is absolutely exempt, and I am only speaking of the seats of selection in the majority of cases. It may also affect the mucous membranes. A considerable number of skin diseases affect the mucous membranes. One of the most important of these is Lichen planus. That also I shall be able to show you to some extent in a living patient. Sometimes on the tongue it forms white patches, and the lesions look like streaks, as if one had put paint on with a brush. Sometimes the patches are in striæ on the buccal mucous membrane and on the lips. These become of importance in diagnosis, for they may precede the eruption on the body. Some little time ago I was called in consultation with a throat specialist with regard to a case of this kind. It was very extensive on the inside of the mouth, going back to the palate, and the question arose, what was the nature of it? It was not so typical as some of these cases, but I affirmed that it was Lichen planus, though she had no eruption on the body at the time. A year later she came to me with very marked Lichen planus on various parts of the body, while the mucous membrane condition was practically unchanged. It is often very obstinate there, but fortunately, it does not give rise to great inconvenience. It may also be seen on the penis and on the vulva. It does not affect the eye in any way. There is a very important feature in the development of Lichen planus. In a very large proportion of the cases in a first attack you can trace that it began in a primary patch. This is a point which has not been generally recognised, but it is one which I have observed for some time; if you can get the earliest stage you find it begins in one point, and may remain there a considerable time, for weeks or months, and occasionally even for years, and then it becomes generalised to a greater or less extent. This peculiarity of starting from a primary patch was first observed especially in a disease called Pityriasis rosea. It was of acute development, where a single patch comes on the body, and after a week or ten days the eruption begins to generalise. You can also often see it in psoriasis, especially when the disease is local to begin with, becoming generalised afterwards. But that is only true of the first attack. Subsequent attacks break out symmetrically. This feature of commencing locally and generalising later is a very strong reason for the early treatment of skin diseases, and it shows that simply because a patch does not give rise to any great inconvenience that is not a reason for it not being treated, while if the first patch is vigorously treated and removed it will take away the danger of subsequent developments.

A vast number of the subacute cases have only the distribution which I have spoken of. But in acute cases it often becomes almost universal, though not absolutely universal like Lichen acuminatus. The face is very rarely attacked and the scalp still more rarely. Still, it may be very widespread indeed. The limbs of this patient were not affected when this drawing was taken, but subsequently they were, and the papules have different characters from the chronic forms. Instead of being of a violaceous tint, they are a very bright red, and are usually very minute and very closely set together. They form large sheets of eruption over the whole trunk and the greater part of the limbs. Some of these acute cases are attended with very severe symptoms, great mental depression, extreme itching, and these, combined with sleeplessness, result in a breakdown of the

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nervous system, but these severe and acute cases are comparatively rare. For every case of this sort, one will see fifty of the subacute. Itching is more or less a symptom in the majority of cases of Lichen planus. In a few the itching is very trifling, but in others it is very severe indeed, and the more the patient scratches the more the eruption is likely to develop in the lines I have spoken of. Then you may get certain complications, though the condition, as a rule, is a dry one. Occasionally you will see bullæ appear, why or wherefore we are unable to say. They are generally few, and mostly upon the lower extremities, but occasionally they are very abundant, and in some cases have masked the disease, as in a case of Kaposi's, in which they occurred at an early stage, and then would naturally make one imagine that actual pemphigus is developing. But that is very exceptional. But to have a few bullæ on the lower extremities is not very rare, and need not shake your diagnosis. Then, according to the anatomical situation of the papules in the skin we get certain difficulties. An ordinary papule develops round the sweat-duct, and you see, therefore, in the middle of a large number of Lichen planus papules a slight horny punctum or a depression. In the drawing I show you of a microscopic section there is a sweat-duct running down the middle, and there is a horny thickening in the centre. When that thickened speck gets thrown off there is a depression. Although that is the rule, and is probably true of all the plane papules, like everything else in medicine, there are exceptions, and occasionally you may get more or less acuminate papules. When that occurs the papule is round a hair-follicle, and in these drawings you will see the anatomical differences between the sweat-pore papule and the hair-follicle papule. These hair-follicle papules are particularly common upon the lower limbs, and they are often associated with a warty condition, and a very common place for it is upon the lower third of the leg. In a less common chronic condition there is a distinct warty appearance. In a similar case to this a papule was excised, of which you see the drawing. These warty cases sometimes give rise to some difficulties in diagnosis, because they are either the remains of a Lichen planus which has cleared up or they are the first manifestations of it. The papule is not, therefore, in strong evidence, and often requires to be carefully searched for in order to demonstrate the nature of the disease. Here, again, that violaceous tint of which I have spoken will be of assistance. Wherever you see a violet patch like that on the leg, whether it is horny or warty, think of Lichen planus, and if you search all the various places in which Lichen planus is likely to develop you will be fairly sure to find some typical papules, although they may be very few in number. Again there are always disagreeable little exceptions to rules. Occasionally you find convex papules, as a rule mixed up with the plane ones. I have seen the whole body covered with little miliary convex papules. A more common condition is a moderate number of larger convex papules, interspersed among the plane ones, which has been called Lichen planus obtusus. The name is not important. So the mere fact of finding a few, or even in the rarer instances a considerable number, of these papules need not shake your diagnosis. But in the vast majority you will find some flat papules if you look carefully for them. There was another rather rare exception in which the papules, instead of being as I have described, are whitish and horny-looking, and if they are aggregated into a group they look very much like that form of sclerodermia which is called morphœa. But instead of the ordinary condition of morphœa with smoothness you will find horny points about it. And then, again, you can often find some of the typical papules in other parts. The reason of the pallor which you see here is that the papule is more deeply seated than usual, and there is some development of fibrous tissue which has obliterated the most superficial vessels. This morphœic form is more of a dermatological curiosity than a very important practical variety. Another pitfall in diagnosis is the infantile form of Lichen planus. That is really not Lichen planus at all. In many cases of miliaria in infants the upper part of the papule scales off and leaves a flat, slightly angular, papule behind it. It is very transitory, and is only what may be called the last stage of a miliary papule. At the same time, true Lichen planus may occur in very young children. I think two years of age is the youngest I have seen, but there are a few younger even than that on record. It is not common till after five years, but it may be seen at any age after that.

What is the etiology of Lichen planus? In this, as in a large number of skin eruptions, some cases are explicable, but in a great many the

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cause is inexplicable. The more slowly developing cases come on insidiously; they do not attract the patient's notice, they may have been there for a considerable time, and the circumstances of the development are so remote that they have been forgotten. At the time the patient presents himself there may be no defect in general health to assist you, for very often the primary causes are removed, though the after-effects persist for a considerable time. This is true of a large number of skin diseases. The mere fact, therefore, that the patient is in perfect health at the time he presents himself does not show that there may not have been some defect at the beginning. Nevertheless, while the eruption is consistent with good health in a great number of cases, in others we can trace nerve-strain as a predisposing cause. The nervous system does not seem to be able to produce any definite skin eruptions, except such as Herpes zoster, but an upset of the nervous system is a predisposing cause to many skin affections. The nervous system is a protector, and if its protection is withdrawn, either from nerve-exhaustion or from toxic causes, or anything which leaves the patient vulnerable, a skin disease may result, but the character of the eruption varies with the individual. We find this disease in patients who have undergone great anxiety and worry, as in those who have nursed sick relatives; in fact, one finds it in overworked nurses themselves, in whom the nervous system breaks down, and an attack of Lichen planus not infrequently follows.

Another cause, and one which I believe is frequently overlooked, is intestinal toxins. People with defective digestions, especially with gastro-intestinal catarrh, are very liable to absorb toxins from the intestine, which produce ill effects upon the skin, and one of those ill effects is Lichen planus. I am one of those who still believe—although it is sometimes ridiculed—that chills can cause disease of the skin, and I think they do it indirectly in many cases, in this way: when the surface of the skin is chilled suddenly, blood is driven from the skin to the viscera, and if there are toxins in the intestine or other viscera, the blood returns laden with those toxins to the skin, and sets up various eruptions. Erythemata proverbially occur in connection with intestinal toxins, and the more subacute forms of Lichen planus, I am convinced, often owe their origin to the same cause. Some of the most acute cases have been definitely connected with chills, and I well recollect one of the most acute and extensive of my cases of Lichen planus occurred in a football player of considerable note, who, there was reason to believe, caught a chill, which was closely followed by this disease. Previously to that his health was perfect, and there was no reason to presume that some constitutional defect underlay the condition and predisposed to it. It is possible, of course, that a great number of even more slowly developing eruptions are connected with such conditions, but they are extremely difficult to trace, and in a large proportion of the more slowly developing cases of Lichen planus we are unable to find a cause for the condition in any particular individual.

Now with regard to the treatment of Lichen planus and the course which it runs. The subacute cases run a very slow course indeed. When improvement takes place it is seen in the diminution in the projection of the lesions, which become less prominent, their colour perhaps becomes paler to some degree, and ultimately the papules sink down even below the level of the normal skin, forming a slight atrophic pit. And this slight atrophic pit is associated with marked pigmentation. Pigmentation in Lichen planus is sometimes extremely deep, a very dark sepia, and this gives rise to errors of diagnosis by those who always associate the idea of pigmentation with syphilis. Of course a large proportion of syphilides do produce pigmentation, but syphilis is by no means alone in that respect, and pigmentation should never lead you to the diagnosis of an eruption as specific. Especially is this the case with Lichen planus. The pigmentation of Lichen planus is, as a rule, deeper than you find in an average syphilitic eruption. The pitting and slight atrophy is, as a rule, only transitory; it does not go below the papillary layer, and all lesions of that kind are recovered from. But in a few instances it does go a little deeper, and then there is permanent pitting left. Still, that is quite exceptional. There are, however, some of the warty lesions which have been of very long standing, and of which the cell infiltration is, of course, much deeper, where atrophic scarring is much more common; but it is rare for individual papules to leave indelible pits. Another feature is that

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occasionally you get a horny punctum in the middle of the papule. Occasionally that horniness persists after the papule has involuted, and you may get a nutmeg-grater-like appearance similar to that of Lichen spinulosus left after Lichen planus; but it is easily removed with soft-soap frictions.

The treatment, as a rule, is on the lines of a psoriasis. That is, you use the same kind of drugs locally at all events; for the same drugs influence Lichen planus as influence psoriasis in many cases, but they require to be used far weaker than in an average case of psoriasis. The first and most important indication is to endeavour to allay the itching. The more the patient scratches the more he keeps the eruption at its full development, and the more additional eruption he brings out. But it is absolutely useless, as many of you know by experience, to preach to a patient that he must not scratch unless you give him something to help him. It is practically impossible when the itching is extreme to abstain from scratching unless some means are devised to give the patient temporary relief. One of the most useful means, I find, is the inunction of oil, with which is mixed a small quantity of thymol, three to five grains of thymol to the ounce of olive oil, rubbed in with pressure rather than with friction. You know there are two ways of applying lotions and oils: one is to press the material in and the other is to rub it in. For Lichen planus in the acute form it is much better for the application to be pressed in, while in the subacute it is, perhaps, better to rub it in. This weak thymol oil generally gives great temporary relief to the patient. Sometimes you will find small quantities of menthol a great advantage.

With regard to the internal treatment for the acute cases the best remedy I have found for most of them is salicin. Here is a drawing of a very acute case which was associated with an unusual condition. He had profuse hyperidrosis of the palms, and some thickening of the horny layers, so that his hands were covered by a thick, horny plate. You can see a purplish eruption on his wrists and hands and practically all over his body. This patient was treated with salicin, and in a few weeks the whole of this horny layer shelled off, and the eruption underwent complete involution, and the patient, from a position of great distress, got into a haven of rest. Another plan of treatment for these widespread generalised cases is an effervescing potash mixture containing a large quantity of quinine. This mixture is useful in a large number of acute widespread inflammations of the skin. The bicarbonate of potash is put in one bottle and quinine dissolved by the citric acid in another. You can give as much as five grains of quinine in that form three times a day without upsetting the patient, if you keep the bowels regular. In the more subacute cases, if it is fairly widespread, salicin is again useful. Arsenic has a great reputation for this, as for many other conditions, but I have myself often been disappointed with it. Another remedy which is of use in a certain proportion of cases is biniodide of mercury. The time-honoured iodide of potassium with perchloride of mercury, which is so frequently given in the tertiary stage of syphilis, is very useful in certain cases of Lichen planus. You do not always succeed with it; nothing always succeeds in medicine. But it does succeed in a certain number of cases, so it is worth trying. In cases where the health is broken down by overwork or by anxiety Tilbury Fox, who was my teacher, and whom I succeeded here, used to give mineral acids with nux vomica. That combination is certainly very useful, and seems to be the best treatment for the right kind of cases. But if there are digestive derangements, or any reason to believe that intestinal toxins are playing a part, you must not be led away with the idea that specifics are useful, but you must treat the patient according to his condition, treat his digestive derangements, keep the alimentary canal clear, give intestinal disinfectants, and in fact let your treatment be on general principles of medicine, and not by specifics. Specifics, of course, are useful and often necessary, but in a large proportion of instances our knowledge of them is only empirical; they are, as it were, a refuge of the destitute, a confession that we do not completely understand the ins and outs of the disease, and therefore we fall back upon these drugs as something which has been found useful, though we do not know why. We do not always tell our patients all that, and I am at the present moment betraying secrets to a layman who has been kind enough to come here to-day to show himself to you. He has a typical Lichen planus. He came to consult me yesterday, and I said to him, "I am going to give a lecture

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on your disease, and you are just the sort of case I should like to show." He at once volunteered to come and show himself. He has papules in the typical position, with one or two of the circles, which are not so common, though I have another case which shows the circles even more marked. This one shows the tint and, in fact, all the characters of the Lichen planus papules better than any case which I have at the present time. Perhaps you do not see these circles more than once in every ten cases of Lichen planus. You can see some of the papules individually and grouped, and you should look carefully with a lens to see their shape, which are seen to be mostly angular and of a violaceous tint. In some of them I have no doubt you will find a pit. He has not so much of the disease on the lower extremities as some have. Nature does not always make these things on one pattern. On the legs there often is a thickened and warty condition and more scaliness. The individual papules are perfectly smooth, but when they are in patches the area is scaly, and sometimes markedly so. The scales are very decidedly adherent. I ask you to notice the shining surface, the purplish tint, and the angularity of many of these papules. He has also a small patch on the mucous membrane of the mouth. Beware of mistaking these conditions of the mouth for syphilitic disease. Sometimes on the buccal mucous membrane they have the form of striæ, like the skeleton of a leaf. This patient has been under treatment and is improving.

This next, a hospital patient, has both syphilis and Lichen planus. The Lichen planus is old and his syphilis is new. He has had his syphilis about two months. Here, again, there is violaceous tint on the legs, which should suggest Lichen planus, whether in a lesion by itself or in association with other lesions. He has also some Lichen planus on the penis, but there is a partially healed chancre there also.

The next patient is a woman, who shows the ring condition very well. On the wrist the papules are almost level with the skin. The rings are very marked on her thighs, there is here also the same purplish tint. But her lesions are much less prominent than they were. At one time they projected more than this. She has been under our treatment some time—that is to say, nine months. She says she has lost the condition several times, but it has recurred. Lichen planus is a disease which is liable to recur, but not with the frequency and constancy of psoriasis. You will frequently find patients who will tell you that they have had previous attacks.

June 18th, 1906.

A CLINICAL LECTURE

ON

DIFFICULT MICTURITION AND RETENTION OF URINE IN THE MALE.*

By JOHN PARDOE, M.B., F.R.C.S.,
Assistant Surgeon to St. Peter's Hospital.

GENTLEMEN,—The subject with which I propose to deal this afternoon is very large, and were I to attempt to follow all the issues to which it gives rise I should do scant justice to a large number of questions, all interesting and worthy of full treatment, and, I fear, leave in your minds but a confused impression of them all. I shall not, therefore, give you a summary of text-book opinions, but shall describe to you the way in which I am accustomed to approach these cases, the methods I use for their relief, the results of my own every-day experience; and if this mode of imparting knowledge to others is of necessity somewhat egotistical, my apology is that it seems to me more alive, and I hope more interesting, than a didactic lecture. I will first draw your attention to what I believe to be the proper way of entering upon the case of a patient coming to you with a complaint of difficulty or inability to pass his urine.

The examination of a patient.—The history is of importance chiefly from the point of view of the duration of the difficulty—the chronicity or the acute onset of the trouble—and it is well to remember that an apparently acute attack of retention is often only the culmination of a long-standing difficulty. In cases of retention it is of the greatest importance to know whether the condition is acute or chronic, for the treatment to be adopted is totally different in the one case and in the other.

You will, therefore, do well to inquire carefully as to the first appearance of symptoms and the course of the trouble from that time until the patient first comes before you.

Other symptoms may come out in the course of your cross-examination, but symptoms are of the most misleading character in diseases of the urinary

* Delivered at the Medical Graduates' College and Polyclinic.

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organs and I would caution you against placing too much reliance upon them.

Two or three instances will serve to substantiate this statement.

Pain at the close of micturition with intermittent hæmaturia and sudden stoppage of the stream of urine from time to time have been looked upon from time immemorial as indicative of stone in the bladder. This particular conjunction of symptoms is, however, seen perfectly in cases of villous growth of the bladder when the pedicle is sufficiently long and the attachment of it sufficiently far forward to allow the growth to engage in the internal meatus. Take another fallacy. Pain in the renal area, referred thence to the groin and testicle, with or without hæmorrhage, leads one to suspect renal stone or growth, yet I have seen more than one case where these symptoms were due to the drag of a bladder-growth upon one ureteric orifice causing reflex pain in the corresponding kidney. The difficulty of diagnosis is still further enhanced in such a case when the ureteric orifice is so dragged upon as to cause partial obstruction to the outflow of urine, and a tense hydronephrosis results. Here all the symptoms of renal trouble are present. I have seen two such cases where a renal operation was averted only by the fact that I take good care to cystoscope as a routine measure of diagnosis.

However, I am wandering from my subject, but I have said enough to emphasise the fact that symptoms are apt to be most misleading in urinary surgery. We must, then, trust rather to physical signs, and the observation of them should be undertaken in a systematic way in all cases of retention of urine.

Examine the patient's general condition first of all, note the condition of the mouth and tongue, for these give a very useful indication in cases of long-standing retention and difficulty. A dry mouth with a somewhat dry, red tongue and continuous thirst means renal inadequacy, due to long-standing back-pressure, the importance of which I shall touch upon presently. Associated with this dry condition of the mouth is often a dry, rough state of the skin, and these patients will tell you that they have almost ceased to sweat.

Next examine the abdomen in the usual way. The regions to which our attention should be more particularly directed are, of course, the loins and the hypogastrium; tenderness and rigidity in the lumbar and iliac regions, even without the actual evidence of a palpable swelling, are suspicious of secondary trouble in the kidneys. Great distension of the bladder is usually evident, palpable and percussible in the hypogastric area. Here, however, I would introduce another note of warning. The bladder is sometimes distended half way to the umbilicus, and yet there is a resonant percussion note right down to the pubes. This is due to the fact that in some people there is a very low reflexion of the parietal peritoneum, and when these patients are fat and the intestines come well down loaded with fat there are sometimes coils of intestine in front of the over-distended bladder which quite mask the usual dulness to a superficial percussion. Deep percussion will, however, bring out the dull note of distended bladder.

Next examine the external genitals, the perineum, and lastly the prostate and parts of the bladder accessible by the rectum.

This routine examination having been carried out, it is time to examine the urethral canal, but this should always be reserved to the last.

In cases where the foregoing examination has shown no obvious signs of disease never forget to examine the nerve-reflexes—knee-jerks, plantar and ocular reflexes—for in a good many cases of spinal cord disease bladder troubles are marked before the nervous condition is very apparent.

Retention of urine.—Patients presenting themselves to you for the first time with a history of being unable to pass urine are, of course, acute so far as the immediate necessities of the case are concerned, but be quite sure to find out whether the case is really an acute one, or whether it is the last phase in a long history of increasing difficulty.

Of the former type I may instance obstruction due to inflammatory swelling in gonorrhœa, or the impaction of a calculus so large as to block the stream, or the retention after operations about the anus or genital organs, and the acute retention so commonly seen by house-surgeons about six o'clock in the morning, when a man with very slight stricture has gone to sleep the previous night full of alcohol and found himself quite unable to pass urine when he arose, more or less sober, in the morning. All these are cases of what I may call true acute retention.

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Far different is the condition when a stricture has been slowly contracting for months or years, or a prostate has been gradually enlarging and obstructing the outflow, or nervous disease has interfered with the free discharge of urine. Here there has been continually increasing difficulty and the flow has dwindled from a stream to a dribble, and finally complete retention occurs. In these cases the bladder has been only partly emptied for months, and after a while in many of them only the excess over a practically full distension has come away by a nearly continuous dribbling, the condition being known as false incontinence or retention with overflow.

Consider now the condition of the urinary organs in such cases.

For many months or years the patient has been straining to overcome his obstacle to micturition, and three conditions or a combination of them result.

(1) The bladder wall gives way generally. The muscular coat weakens and thins out, and the bladder is distended more and more.

(2) The backward pressure does not appear to be exercised so much upon the bladder as a whole as upon the ureteric orifices. The ureters dilate, the pelves of the kidneys dilate, finally the cortical kidney substance is spread out as the thin wall of a tense hydronephrosis.

(3) The bladder muscle first hypertrophies to meet the increased tax upon its power, but eventually the hypertrophied muscle bundles separate and allow the gradual protrusion outwards of the mucous membrane, resulting at last in the condition of fasciculated bladder, or as the French expressively describe it, *vessie à colonnes*, the final stage of which is the pouched or sacculated bladder.

Now, all these stages are finally accompanied by a serious back-pressure upon the kidneys, and when the condition of back-pressure is very marked the kidneys have to do their work against a pressure I was almost going to say higher than that of the blood. I cannot go so far as that, for I do not know if the blood-pressure and the intra-vesical pressure in these cases have been tested, but at all events they have to work at a very grievous disadvantage. The cortical tissue is thinned out and compressed by the immense dilatation and the blood has poor access to the glomerular tufts.

The formation of urine, as you will remember, is not only a filtration, but it is also a true secretion. This process is much impaired as evidenced by the marked diminution in urea and the great lowering of the specific gravity of the urine in these cases.

Therefore, gentlemen, when a patient comes to you with a history of long-standing difficulty, with a urine of very low specific gravity, with thirst, lumbar aching, dry tongue, and harsh skin, be very wary how you deal with him instrumentally, for these signs mean renal inadequacy, due to back-pressure.

Not a year passes without the medical Journals recording some such case as the following imaginary one:

A patient, sometimes a prostatic case, sometimes a woman with retroverted gravid uterus, sometimes a man the subject of long-standing stricture, presents him or herself with a bladder well distended above the pubes. The doctor passes a catheter and there and then draws off a very large amount of urine, not seldom measured in quarts. Within a few hours the patient is very ill, with some fever, much lumbar pain, and complete or almost complete suppression of urine, and he dies in a few days with all the symptoms of uræmia. In some cases the shock is so extreme that the patient succumbs in a very few hours.

Now, all these cases are marked by the same features, viz. long-standing difficulty and retention, sudden withdrawal of a very large amount of pale limpid urine (the point which is usually rather proudly insisted upon), and the speedy death of the unfortunate sufferer.

What is the explanation of these phenomena? First, in those cases where fatal symptoms of shock are the main feature I think they are comparable to the sudden withdrawal of large amounts of fluid from the abdominal cavity in cases of ascites or large ovarian fluid cysts, with the added fact that there is undoubtedly a curious tendency to shock on instrumental interference with the urinary organs.

But by far more important is the effect upon the kidneys. They have been thinned out by a long-continued centrifugal pressure, and they are of necessity intensely anæmic. This force is suddenly removed, the balance sways down heavily, and these thinned out, starved, anæmic kidneys suddenly have to bear a great inrush of blood.

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They become intensely engorged, and post mortem are sometimes found almost purple in colour, with small hæmorrhages everywhere visible. There is no wonder, then, that these patients die.

All this somewhat prolix explanation leads up to the axiom which I wish to impress upon you this afternoon, and that is, never under any circumstances, however much the patient may urge you, suddenly empty a chronically over-distended bladder. This point has not been sufficently insisted upon in text-books of surgery and gynæcology, and when mentioned at all it is not emphasised.

In these cases you should take several hours to empty the bladder, and this is very easily done either by continuous or intermittent drainage. In the former case a small catheter is tied in, so small that the urine only escapes in a slow dribble or drop by drop, and you will find that in twenty-four or forty-eight hours the bladder is empty. This is the better way, but if your patient will not consent to this a small catheter can be inserted every four or six hours and rather more drawn off than the kidneys secrete in the intervals.

In any case your patient must be put to bed, with warm bottles, warm, easily digested nourishment at frequent intervals, and such things as lemon-juice, lime-juice, etc., given to quench the thirst, with the prompt exhibition of stimulants if need arises.

This serious condition of bladder and kidneys which I have described to you is the outcome of long-standing obstruction only, and is never present in cases of true acute retention. In such cases, then, there is no need for these precautions. The full bladder may be emptied at once, with little or no fear of these dangerous consequences, and I trust you clearly follow the distinction between the two types.

The methods of relieving retention.—Let us now turn to the practical consideration of the methods of relieving retention of urine. I premise first of all that we have tried the useful method of giving the patient a full dose of opium and seating him in a warm bath for twenty minutes or half an hour, if his condition warrants the latter measure. In any case it is a good preliminary whilst we prepare our instruments. There are two methods of emptying a bladder, first by means of urethral instruments and secondly by direct puncture. Let us consider the latter first.

Puncture through the rectum and Cock's perineal puncture, I think, are dead and done with, fortunately. There remains suprapubic puncture. This is the simplest little operation imaginable, but, like most operations, it can be done in a right and a wrong way. First make sure that you have no bowel in the way by careful percussion. Make a small nick in the skin just above the pubes, which has been shaved and cleansed, and then plunge firmly downwards and a little forwards (the patient lying on his back) a medium-sized aspirating needle, which can be attached to an aspirating bottle. Then draw off the urine slowly. Syringe a little boracic fluid through the needle previous to withdrawal to clear it of urine and seal the small nick in the skin with collodion. If such instruments as these are not at hand, a small but long hydrocele trocar will do perfectly well. This little operation can be repeated if necessary, but if the bladder is emptied once time is obtained to deal more radically with the obstruction. There is just the one danger in connection with this method, that of wounding the bowel. If there is any doubt about the bowel being in the way, I prefer to make a finger-hole incision and pull up the peritoneum with the finger until the bladder can be seen, thus avoiding all risk.

Supra-pubic puncture is but very rarely called for if—and it is a large "if"—the practitioner has a proper selection of urethral instruments at his command.

But there is one condition where supra-pubic puncture is extensively advocated as the first means of relief to be employed, namely retention of urine due to gonorrhœal inflammation.

In my experience this is an extremely rare complication of acute gonorrhœa, and this is not surprising when we consider that in the great majority of cases the infection is limited to the anterior urethra. But when the posterior urethra is infected and considerable swelling occurs retention does occasionally take place.

Even in these cases surgical interference is very rarely called for, the retention being relieved by hot hip baths, free purgation, and the use of opium in reasonable doses.

But if all palliative treatment fails and the retention persists there are those who say that a catheter should never be passed, but that relief should always be gained by supra-pubic puncture

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for fear of infecting the bladder if the urethral route is chosen. But if reasonable precautions are taken I think the danger of bladder infection is rather over-estimated in these cases. When we consider that the membrano-prostatic urethra is infected in almost all instances of gonorrhœal retention, and that the infection is in the epithelial cells themselves, the epithelium being directly continuous with that of the bladder, it seems rather unlikely that a catheter passed once or twice with proper precautions will cause any more infection than that which might occur by direct extension of the process along the mucous membrane.

Those of you who were present when I delivered two lectures last autumn on the treatment of chronic urethritis will remember that I described to you Janet's method of posterior urethral irrigation, which is extensively used by some surgeons even in subacute cases. By this method the contents of the posterior urethra are necessarily swept back into the bladder, and although infection of the bladder does undoubtedly occur sometimes, it is a very rare occurrence indeed. My own practice in these cases is to carefully wash out the whole anterior urethra, and then to inject into the bulb and posterior urethra a solution with the following formula:

℞. Eucaine B. . . . gr. x.
Adrenalin hydrochlor. . ℥ij.
Aq. destillat. . . ad. ℥j.

Thirty to forty mimims of this solution should be introduced with a Guyon's syringe, and allowed to remain in the urethra for ten minutes or more. The effect is to relieve pain and thereby relieve spasm, and, in addition, a temporary reduction of swelling is also caused. In more than one case that I have seen the urine was passed naturally at the end of ten minutes without the aid of any instrument.

But if after waiting ten or twenty minutes the retention still persists, then I think that a small catheter, very gently passed, is the cause of very little danger and may be repeated once or twice. These retentions seldom last more than forty-eight hours, and four insertions of the catheter would be the utmost called for.

This is a debatable point, however, and I am merely giving you my own opinion upon the matter. But please remember that the anterior urethra must be most carefully washed out prior to the introduction of the catheter, and that the adrenalin eucaine solution should first be injected.

Other conditions calling for supra-pubic puncture may be briefly mentioned. Occasionally a series of difficult strictures is complicated by an enlarged prostate. The surgeon is able to negotiate the strictures, but his instrument being tightly gripped in them, he has no command over it when it reaches the prostate and he cannot pass this obstruction. Here the obvious thing to do is to give relief by supra-pubic puncture and thus gain time and relieve tension preparatory to further gentle efforts.

Retention due to a large malignant prostate where catheterism causes severe bleeding may be relieved by supra-pubic puncture prior to permanent supra-pubic drainage. To sum up, it is a useful little operation as a means of gaining time and relieving urgent symptoms preparatory to more radical treatment.

Relief of retention by urethral instruments.—Having carried out your routine examination of the patient in the way I have described to you, let us assume that the case is one of stricture, and of tight stricture.

First cocainise or eucainise the urethra by injecting twenty or thirty minims of the solution for which I gave you the formula. This can be done with a small glass syringe or glass pipette with rubber teat. Holding the injection in, strip it down towards the bulb by pressure with your fingers and let it remain for three or four minutes. Then take a fine silk-web French bougie and pass it very gently down to the stricture. The calibre of the bougie should be about No. 4 (French scale). If it refuses to pass try smaller instruments down to a filiform, and whatever instrument you get through my advice to you is to leave it in and let the urine dribble alongside it. But if the stricture is not so tight and difficult you will find that one of these small French bougies will slip through fairly easily. In such a case you can continue in series with bougies up to Nos. 9 or 10 (French scale) and follow this with an olive-pointed French catheter two sizes smaller than the largest bougie you have passed. Then tie this catheter into the bladder, and leave it there for twenty-four hours. At the end of that period you will find that you can proceed further with the dilatation.

Some of the finer instruments are made with a

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small bend at the tip and others with a spiral of one or two half turns. The latter are called corkscrew bougies, and are sometimes very useful to hit off the opening of a stricture.

But if the passage of your instrument is not arrested until it has passed beyond the bulb, you are then dealing most probably with enlargement of the prostate—and you will remember that a prostate which feels quite small *per rectum* may cause as much and more obstruction than one which feels very large. In such cases your very small instruments are not necessary. Choose a silk-web blunt-pointed Coudé catheter, such as I show you here, about No. 8 or No. 10 (French scale) and pass it gently down to the prostate. Always pull the penis well up on the catheter with the left hand. You are better able to feel what the point of the catheter is doing if you do this, and always remember to keep the bend of the catheter upwards, the object of the bend being to ride over a median obstruction if one is present. If you find that the catheter is stopped, a forefinger in the rectum pressing against the instrument in the prostatic urethra, whilst the onward pressure is continued with the other hand often helps matters considerably. The manœuvre just gives the requisite tilt to the point of the instrument, and it passes the obstruction.

Here I show you a very useful instrument in these cases. It is called a whip catheter and was designed by Mr. Reginald Harrison. You will notice that the point is very fine, long, and flexible, and this enables it to wriggle round corners and over projections, finding its own way so to speak. For five or six inches the instrument is a solid bougie, but as it grows larger you will notice that it is hollow and there is an eye upon the side. The long flexible end curls up in the bladder as you introduce the instrument and the urine flows so soon as the eye enters the bladder. The same end, viz. that the instrument should find its own way, is sometimes attained by the use of a Jacques' rubber catheter. Always use those catheters which are provided with what is called a solid end and a velvet eye. The catheters which continue hollow beyond the eye leave a *cul de sac* difficult to clean after use and should be avoided.

Sometimes, but very rarely, a soft instrument will not pass in these prostatic cases. You may then try very gently to introduce a full-curved metal catheter, but no force whatever must be used.

Another useful instrument is the flexible catheter, either gum-elastic or varnished silk-web fitted with a stout metal stylet. Give to the catheter with the stylet in it the full curve which you desire. Pass the instrument thus armed into the prostatic urethra and then withdraw the stylet for an inch or two. This leaves the point of the catheter flexible whilst the rest of it is rigid, and very often you can negotiate the obstacle in this way.

I see that I have already overstepped the bounds of time, gentlemen, and have not yet entered upon a consideration of some points about which I hoped to speak to you this afternoon—for instance, the treatment of obstruction to micturition by foreign bodies in the urethra, the routine treatment of stricture, with the choice of cases for dilatation and operation respectively, the treatment of inflammatory conditions of the prostate obstructing micturition, and so forth. I shall hope to deal with these and other branches of this subject on a future occasion, but before you go you may like to see what I consider a full outfit of instruments which will enable any practitioner to deal adequately with any case of retention which may arise. You will notice that the list is not a long one nor is it very expensive.

[Mr. Pardoe showed the members of the class the selection of instruments and demonstrated their suitability to various types of cases.]

June 18th, 1906.

Rollin says that while the older clinicians were accustomed to say that the mucous membrane of the tongue mirrored the conditions existing in the stomach, modern teaching has discredited this view. He maintains, however, that there is a good deal of truth in the former opinion, and that it is incorrect to assume that no immediate connection can exist between gastric disorders and the state of the tongue. Observations on the fasting stomach contents in cases of hyperacidity and of subacidity show that while in the former condition a great deal of lingual epithelium is present, in the latter affection the reverse is true. In hyperacidity the tongue is nearly always clean, whereas in subacidity it is proverbially coated.—*Med. Rec.*, vol. lxix, No. 21.

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A CLINICAL LECTURE ON CAISSON DISEASE.

Delivered at St. Bartholomew's Hospital.

By HOWARD H. TOOTH, C.M.G., M.D., F.R.C.P., Physician to the Hospital.

GENTLEMEN,—Some of you may remember that some time ago, in Matthew Ward, there was a patient, an underground worker, who was suffering from certain symptoms which are grouped under the term "caisson disease" or "compressed air illness." And I thought that case might serve as a text for some remarks on the general subject, because it has become an important one in consequence of the large amount of engineering works which require to be executed at considerable depths. It is therefore a condition which you are not unlikely to be called upon to face in practice in after-life.

The patient was a man æt. 44 years, who was admitted on December 13th, 1905. During the last sixteen years he has been working at intervals in caissons under compressed air, in all, on eleven different jobs. But the pressure under which he has had to work has not been more than fifteen pounds to the square inch—that is to say, + 1 atmosphere. During that period he says he experienced no ill effects except occasional epistaxis. In April, 1905, he was at work on the new "tube" railway from Hampstead to Charing Cross, and while on that work they came upon a water-bearing stratum somewhere under Maple's establishment in Tottenham Court Road. This necessitated the use of compressed air at twenty-five pounds to the square inch. After he had worked at this pressure for two or three shifts of eight hours each he began to feel symptoms. He says he had symptoms both in the caisson and afterwards. While under compression at work he had occa-

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sional symptoms, such as deafness in the right ear, epistaxis, and bleeding from piles. Fourteen days before finally giving up work he suffered from symptoms on leaving the caisson. These symptoms were particularly pains about the joints—that is to say, shoulders, elbows, knees, and ankles—and these pains were very severe and almost continuous, but with occasional exacerbations. He also had sensations about the joints, suggesting, he said, "fluttering birds." He frequently had humming in the ears, and he sometimes became so weak in the legs that he was almost unable to walk at all.

On examination, we found him much reduced in body weight: he said he had lost two stone. He was in a decidedly neurasthenic condition; his mental attitude was introspective; he was most voluble in his description of his sufferings. His temperature was generally subnormal, the tongue slightly coated and tremulous, the eyes unsteady on conjugate deviation, as is commonly found in neurasthenics. The pupils were equal and reacted to light. His hearing was somewhat obtuse, he could not hear the watch farther than six inches away. His grasps were feeble, but equal. Sensation in the hands was delayed, and there was marked hypoalgesia. His gait was unsteady, suggesting functional weakness only; it was not distinctive of any organic condition. The knee-jerks were feeble, the plantar reflex gave the normal flexor response. There was slight hypoalgesia of the lower half of the left leg, but not corresponding to any particular root or nerve distribution. So at this time the case seemed to be one of neurasthenia and came into line with those of caisson disease recorded, only in respect of the pains or "bends" about the joints.

He was treated by tonic medicines and massage, kept in bed, fed well, and in consequence he rapidly put on flesh, and although he complained of pains from time to time we heard less and less of them. Finally he ceased to have them any longer and asked for his discharge, going home on February 1st—that is to say, seven weeks after admission, and then he was practically well. The attacks of pain were much relieved and cut short by antifebrin.

It is rather surprising that we see so few of these cases both in general and special hospitals. I suppose we may assume that the majority of the cases get well fairly rapidly, and also, I hope, that the precautions taken are now so effectual that the more serious cases have become rare.

A somewhat copious literature has grown up around this subject, which, however, is treated very shortly in most of the text-books. References are to be found in the works of Snell,* Parkin,† and Leonard Hill and Macleod;‡ the latter have recorded much valuable experimental work.

The conditions under which compressed air is used are as follows: There is first the diving-bell, which in its earlier form was a hollow vessel without inlet above, but open below, which was lowered into the water, falling by its own weight. As it descended in the water the pressure rose inside, and at the same time the water was driven to a certain distance within the bell, so that when it reached the bed there was always about a foot of water to work through. Of course the ventilation of such a structure was *nil*. A great improvement was the provision of an inlet tube through which air can be pumped, thus providing the occupants with fresh air and at the same time forcing out the water from the mouth of the bell, so enabling the divers to work on a comparatively dry bed. The measure of the pressure within was the depth in feet below the surface of the water, 33 feet being equal to one atmosphere.

Another apparatus in which compressed air is used is the diving suit. The earliest form was open at the legs like a miniature diving bell; air was pumped down and bubbled out at the bottom. The objection to this was that if the man fell down the water was apt to run up into the suit and he was in danger of drowning, therefore the open suit was discarded for the closed one, which is the form now in use.

Lastly, we come to the caisson, derived from the French word *caisse*, a case or box. The caisson is essentially a tube, lowered vertically through the water to the bed of the river or dock at which the work has to be done. Of course in shallow water the water can be kept out by pumping, but this is out of the question when the work has to be done at any great depth. Under these circumstances compressed air becomes a necessity,

* Snell, 'Compressed Air Illness,' 1896.

† Parkin, 'Caisson Disease,' Thesis Durham University, 1905.

‡ Hill and Macleod, 'Proc. Roy. Soc.,' 1902; 'Journ. Physiol.,' 1903, p. 492; 'Journ. of Hygiene,' 1903, p. 401.

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and it is used for all large works, such as the building of piers for bridges—for instance, the St. Louis Bridge and the high-level bridge at Newcastle. Near the bottom of the tube is a partition which cuts off the lower end to form a chamber into which the compressed air is pumped. From this chamber runs a shaft, up and down which the men go and the material also which is removed from the bed of the river. At the top of this shaft is another small chamber, the air-lock, which can be cut off from the shaft by traps or valves. As the pressure rises in the compression-chamber the water is forced out at the bottom and kept out. The quantity of air which is passed in represents, to some extent, the ventilation of the chamber, and the pressure is measured, as in the diving-bell, by the depth at which it is below the surface of the water. The air-lock is an important element in the apparatus. The workman enters the lock, the door is closed, and the air forced in; when the presssre has risen to that of the rest of the system the traps can be opened, and he descends the shaft into the working chamber. This is a rough description of the principle of a caisson, which, in the vertical form, is practically a huge diving-bell. In the tunnelling operations which are now going on so much in London in connection with the various "tube" schemes the compressed air chamber is frequently needed. It was used extensively in the Blackwall Tunnel operations. In this case the caisson, of course, is the tube which ultimately lines the tunnel. A compressed air-chamber becomes necessary in tunnelling when a water-bearing stratum is encountered, or where, as at Blackwall, the tunnel is close under the bed of a river.

I must trouble you with a few figures, but they will be easy to remember. You know that the pressure of the atmosphere in which we live is about 15 lb. to the square inch, and that an atmosphere is taken as the unit of pressure. Put in another way, an atmosphere represents 33 feet of water or a column of mercury 32 inches high. Therefore, if you lower your diving-bell 33 feet below the level of the water you will have raised the pressure of the air within the bell 1 atmosphere, but the actual pressure is 2 atmospheres, or 30 lb. to the square inch. The greatest depth at which, so far as I can gather, work has been carried on is 201 feet. The Admiralty require that their divers shall be able to work at 12 fathoms (= 72 feet) for one hour, 15 fathoms (= 90 feet) for half an hour, and 20 fathoms (= 120 feet) for a quarter of an hour. These times are reduced if the temperature of the water be below 42° F. The caissons which we have described here rarely work at more than 100 feet depth, which is 60 lb. pressure to the square inch. At the St. Louis Bridge, one of the biggest works of the sort, the men worked for eight-hour shifts at a pressure of 4½ atmospheres, and numbers of men suffered badly from the pains or "bends," in consequence of which it became necessary to shorten the shifts and to lengthen the time of decompression.

When the workman enters the air-lock the process of "locking-in" is commenced. There is very little unpleasantness. Sometimes he feels pain in the ears, but that is relieved by swallowing movements. The time taken over this part of the process seems to be a matter of indifference. Snell considers that no harm can come from raising the pressure to 30 lb. to the square inch in one minute. There are certain physiological and physical effects. The air in air-containing viscera—namely, the bowels, the sinuses of the skull and middle ear—becomes compressed and occupies therefore less space, and there is a feeling of emptiness in the region of the stomach, so that the men almost instinctively begin to tighten up their belts; the diaphragm descends more readily, and therefore the breathing capacity of the chest is improved. But it is around certain physical facts connected with the absorption of gases by liquids under pressure that the interest centres. When a man is subjected to air under compression the fluids of his body, the blood principally, conform to the law of absorption of gases known as Dalton's law—namely, that gas, mostly nitrogen, is absorbed directly as the pressure and inversely as the temperature. This absorption must take place through the lungs almost entirely. It must also take time, and the ultimate saturation of the blood and the tissues for a given pressure will therefore be a question of time. A man may go under compression for a short time at intervals with almost complete impunity, but it is a very different matter if he works in, say, 30 lb. of pressure for eight hours. By that time his blood and tissues have absorbed the maximum of gas for that pressure and temperature, and, as Dr. Hill expresses it, he

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resembles a bottle of soda-water charged with gas in solution, and as the gas bubbles out on drawing the cork, so there is a similar tendency for the dissolved gas to escape from the blood on lowering the pressure. This question of the time taken for saturation is one of great importance, and one on which accurate knowledge is needed. Drs. Hill and Macleod ('Journal of Physiol.,' 1903, p. 385) find that it takes a dog of 5 kilogrammes one and a half hours to become saturated, and it is therefore not unreasonable to suppose that it must take a man at least three times as long.

In the St. Louis Bridge works there was a pressure of four and a half atmospheres, and the men suffered considerably from what they called the "bends"—that is to say, pains around the joints. The deeper the working the more frequent became these pains. Therefore the shifts had to be shortened to two hours, and finally one hour. The question involves not only the safety of the workers, but economy in working, which will be more evident on consideration of the decompression or idle period necessary for the safe use of compressed air. There is also a certain amount of loss of body heat, arising from the fact that the air is saturated with moisture. One might expect that the output of nitrogen would be increased with the metabolism, but Dr. Hill finds that this is not so. And the blood-pressure is unaffected in animals subjected to pressures far higher than the labourer is ever expected to work under. I have already mentioned the little discomfort connected with the ears felt on going into the compressed air chamber, relieved by the movements of swallowing, which re-establish the pressures within and without the tympanum. Men who are permanently deaf from closed Eustachian tubes run serious risks of bursting of the tympanic membrane, with hæmorrhage within the tympanum, under these circumstances. When in the compressed air chamber whispering and whistling and talking become difficult, and it becomes necessary to speak more loudly than at atmospheric pressure. But there is a general feeling of well-being among the occupants of the air-chamber, graphically described in the words of a workman in Hill and Macleod's paper ('Journ. of Hygiene,' 1903, p. 405). This may throw a sidelight on the influence of meteorological variations of barometric pressure on certain sensitive nervous organisations in ordinary life. There is no doubt that persons suffering from disease like disseminate sclerosis with much spasticity of muscles are profoundly sensitive to changes of weather.

I now come to a very important part of the question—namely the symptoms which may follow the period of decompression. When the workmen leave the air-chamber they go up into the air-lock, and there they have to remain for a certain time. If the lowering of pressure is made too suddenly, such, for instance, as by the accidental bursting of a caisson, the occupants die in convulsions. It appears to be almost impossible to impress upon the men the importance of gradual decompression; the tendency with them is to hurry this period in order to get away home, and Nemesis in the shape of pains is almost sure to follow.

The symptoms during and after decompression are so numerous and varied that it is difficult to classify them, so I have jotted down some of the principal ones. When we consider the underlying pathological condition, namely escape of gas into the tissues, it is not surprising to find infinite variety in symptoms. Bleeding from the nose, hæmoptysis, hæmatemesis, and melæna, are not uncommon. But the most common symptom is that of pain either in the muscles of the limbs or in the neighbourhood of the joints. These pains are known by the general name of "bends," and it is difficult to picture what they are like from the description given by the patient. Sometimes they are described as "cramps," and in our patient, besides these indescribable pains, there were other sensory symptoms of a subjective nature suggesting to his mind a "fluttering" sensation. These sensory symptoms may owe their existence to local causes, affection of peripheral nerves, or more likely to central posterior root lesions. Aural vertigo is a common symptom; in fact, it is rather a constant one. Sudden attacks of fainting, prolonged into coma, are not very uncommon, possibly due to air-embolism in the brain. From the same probable cause occur temporary paralyses of one limb, hemiplegic in distribution, or even paraplegic, without loss of bladder control. Recovery is generally rapid and complete, but occasionally this may be delayed for some months. So serious a lesion as acute necrosis of the femur, necessitating amputation, has been recorded, probably due to embolism of the medullary artery of the bone.

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It is clear, then, from the foregoing remarks that there is little danger to the individual in entering a chamber under even high compression, or in remaining under that pressure for comparatively long periods, but to avoid symptoms it is necessary to pay close attention to the process of decompression and the time taken in lowering the pressure to that of the atmosphere. Here arises, as it were, an antagonism between the interests of the workman and those of the employer, for the workman must undergo after each shift a longer or shorter period of enforced idleness, irksome to himself and unremunerative to the employer. The longer a man is under compression the more he is saturated with gas, at least, up to his saturation point, whatever that may be and whenever it may occur. If the man is in the compressed air eight hours, as he often is in low pressures, it is probable that he will be completely saturated, according to Dalton's law, for that time and under that pressure. The higher the compression the shorter should be the shift for the men, because the more quickly will they become saturated with the gases, and therefore the longer should be the decompression period.

Hill and MacLeod suggest the following as ideal shifts and decompression periods for the various pressures. Under a 2-atmosphere pressure, or 30 lb. to the square inch, the shift should not be longer than four hours, and the decompression period anything from half an hour to one hour. So that to every four hours' work must be added from a half to an hour's unremunerative leisure, an important item in the wages sheet. In + 3 to + 4 atmospheres pressure—that is to say, 45 to 60 lb. to the square inch—they suggest a four-hour shift, but one to two hours' decompression. For 5 atmospheres, or 75 lb. to the square inch of pressure, the shifts they suggest should be one hour and two hours' decompression—that is to say, the men should spend longer in the decompression chamber than at work—and at very high pressures, 6 to 7 atmospheres, the men should not be at work more than half an hour to one hour, and should have two hours' decompression.

There are certain other points which I must refer to. First, ventilation. Dr. Snell, from his Blackwall Tunnel experience, suggests that the ventilation should amount to 9000 cubic feet. Normally, 3000 cubic feet of air is considered necessary for health, and under hard-working conditions such as I have been describing Snell thinks there should be at least three times as much. That seems a very heavy requirement, and if twenty-five men are at work in a comparatively small chamber to give them 9000 cubic feet each the air must be pumped in at such a rate as to create a great draught, and, consequently, to lead to such a lowering of temperature that it is a question whether the remedy is not worse than the condition which it is meant to alter. Probably in most works the supply of air is nearer 1000 to 2000 cubic feet.

Another condition which these men are subjected to is dampness of the atmosphere, and as the pressure rises a cloud makes its appearance, with the result that the men's clothing becomes saturated with moisture, a condition predisposing to rheumatic troubles.

Dr. Parkin describes how, in the Newcastle Bridge works, they came on a small coal-bearing stratum giving off a quantity of hydrogen sulphide gas, and that while they were on that stratum the men felt the oppression very badly, a point bearing again on the question of ventilation. In the process of filling in the caisson with concrete to form the pile of the bridge gases are given off very noticeable and unpleasant under the conditions in which the men work.

Finally, there is the annoyance caused by the fouling of the caisson by the excreta of the workmen, which should be obviated by the provision of earth closets, frequently emptied and cleansed.

All these conditions must be considered as contributory to the production of the symptoms proper to working under compressed air in small chambers. A very important question from the point of view of prevention is the selection of the men for the work, and these should be no other than men who would be considered first-class lives from an insurance point of view. There would appear to be one important physical condition underlying the varied symptomatology of the so-called caisson disease, namely the solution of gases (nitrogen and oxygen) in the plasma of the blood and the juices of the tissues generally. As stated above, this solution takes place under definite laws. Whilst the individual is under pressure the dissolved gases are harmless. But when the pressure is removed these gases must leave the fluid in which they are dissolved, and unless the process of decompression is very gradual, small bubbles of gas are let loose. These have been seen in animals actually circulating in the capillaries and also free in the tissues—the eye, for instance. A

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sudden appearance of air in the small vessels and capillaries generally must inevitably cause a rapid rise of blood-pressure, and thus put a dangerous strain on the heart; locally such bubbles have the effect of emboli. It is in connection with these facts that serious accidents may occur. The bubbles disseminated in the tissues may cause local surgical emphysema but need not necessarily be dangerous. But a considerable evolution of gas in the chambers of the heart may be of the utmost gravity. It is, again, easy to see that an air-embolism of the cerebral or spinal vessels may cause serious or only temporary paralyses. So also the escape of gas into the spinal cord may possibly account for pains from local pressure on the posterior root zones. On the other hand, it is highly probable that a return to the high-pressure chamber may cause a re-solution of the escaped gases, and this will explain the rapid improvement in the symptoms if this procedure is adopted soon enough. In order to carry out this treatment a "medical air lock" is now provided in large works. It consists of a large metal chamber, such as an old boiler, in which the men showing symptoms can be subjected again to pressure and very slowly decompressed. There is no doubt that the frequent occurrence of pains is an indication that the patient should give up this sort of work entirely.

June 25th, 1906.

A LECTURE

ON

PROGNOSIS IN PULMONARY TUBERCULOSIS.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton.

By H. BATTY SHAW, M.D., F.R.C.P.,
Assistant Physician to the Hospital and to University College Hospital.

GENTLEMEN,—Prognosis in pulmonary tuberculosis may well appear to be an impossible task, or at least one of the most difficult that can be approached. Aufrecht in his recent monograph on 'Pulmonary Tuberculosis' devotes little more than three pages to the subject and adopts a non-committal attitude. The importance of the subject is, however, manifest, for no sooner is the disease discovered than the practical question arises, What plan should be adopted for the future?

The most optimistic physician can but share the layman's view, that tuberculosis of the lungs is a grave disease, although he may feel that the teaching of the post-mortem room encourages the belief that tuberculosis can be cured. Four to twenty-three per cent. of all cases examined post mortem show healed tuberculous lesions in the lungs (Hector Mackenzie), and there are many physicians who can quote examples of patients afflicted with pulmonary tuberculosis who have ultimately apparently become cured, or who have led long and useful lives, though saddled with the nightmare of the dangers of chronic tuberculosis; they can also quote cases of tuberculosis who have died of other diseases. Financial considerations are insistent, and the physician who is asked as to the future of a tuberculous case may well reply with the question, What are the financial resources? for he measures the future largely by considerations of the amount of expense that can be borne by the patient or his friends.

The object of this lecture is to put before you some of the main considerations which may be of prognostic value. Tuberculosis of the lungs is in a large number of cases pre-eminently a chronic disease; "acute cases" of pulmonary tuberculosis other than cases of miliary tuberculosis arouse our suspicions that the cross-examination of the patient as to previous illnesses, which are but forerunners of the disease or are early manifestations of it, is at fault or that our physical examination is unsatisfactory. Too often acute pulmonary tuberculosis means only auto-infection or the final blaze-up of smouldering mischief, the more intense and acute by reason possibly of an exaltation of the virus or of a final depression from many causes, of the individual's protective powers.

To arrive at anything like a satisfactory idea of the future course of a case of pulmonary tuberculosis, attention must be paid to the study:

(1) Of the patient as an individual, bearing in mind his physique, temperament and social surroundings.

(2) Of the particular character of the disease; for tuberculosis of the lungs presents itself in a variety of ways—acute, sub-acute, or chronic, or complicated as in mixed infection or by other diseases, such as diabetes, Bright's disease, etc.

(3) Of our own powers of recognising the evidence of disease, its extent and severity, under a variety of associations which only long practice

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can adequately teach us to appreciate in their full significance.

(1) *The patient.*—It is always a hard task to settle whether the particular temperament shown by a patient is an expression of mildness or severity of the illness or whether it depends entirely upon individual training, education, etc., but it is very generally admitted that highly excitable patients, who take their troubles in full measures of seriousness, frequently defeat all efforts made to successfully treat their disease. Beware therefore of the highly excitable patient, who eagerly watches every expression of your face and asks countless questions as to the future. Phthisiophobia to such people is a grave matter and every effort must be made to show that panic is uncalled for and even harmful. Despite every effort in those cases, however, too often the fear intensifies, and the final scenes soon follow.

It is well for you to form an estimate of your patient quite apart from physical examination of the chest, history, etc. You will find that in some cases your patients look well; they even declare that they feel well, and their behaviour in refusing to avail themselves of special hospital or sanatorium treatment shows that they are not overmastered by the disease, however extensive the signs may be. Their colour is good, they appear to be well nourished, and their outlook is entirely a hopeful one. Such cases will be found as a rule to do extremely well, and form a most marked contrast to the depressed or excitable, anxious individual in whom pallor, emaciation, slack muscular power, dry, lustreless hair are marked features, although chest signs may be at a minimum. This general view of your patient is put first, not only because it is the most important guide as to the future, but because experience has taught us that too often we attend for "examination purposes" to the physical signs, as given by the more technical methods of examination, rather than to the aspect of the patient. No doubt you will be tempted to consider this general survey of the patient an unreliable test, and may be able to quote cases which fared badly despite the favourable impression of a general survey and cases also which looked hopeless and yet with suitable treatment reacted in a most favourable manner. One patient, for instance, to be seen presently was actually refused admission as an in-patient more than a year ago because his general aspect was so poor; he is still alive and as an out-patient has thriven in a remarkable way. Another case is that of a boy æt. 7 years, at University College Hospital, who was admitted with extensive signs of disease in the right lung and with signs of kidney involvement. He has now lost his fever, is gaining weight, and from being considered the most hopeless in the ward has become the "show" case. These exceptions must not blind you, however, to the general truth of the statement that it is most important to study your patient as well as his disease. It is most unfortunate that we have no absolute test at present which is free from error, by which we may estimate this general power of the patient to resist disease, but a movement is already on foot for the estimation of the protective power of the blood which may provide us with a new "arm," not only for prognosis but as a guide in treatment.

Age.—The disease in children is usually very rapid but exceptions do occur. Patients in the second and third decades form the bulk of those who seek relief as out-patients here; often they make only two or three visits, being too ill to present themselves for further treatment. It is a suitable moment to remind you that your study of tuberculosis of the lungs in a general hospital gives you inadequate views of what we know is, as a rule, a most protracted illness. You see cases admitted for pneumothorax or for severe hæmoptysis, but it is impossible for cases to be provided and kept under observation long enough in a general hospital; such patients require nursing, not for a few weeks but for months, and as in other forms of infectious disease such as typhoid, it is fast becoming acknowledged that tuberculous patients should be treated together in special wards or hospitals where everyone is on the alert to prevent infection. It is not very common for elderly men and women to present themselves here for tuberculosis of the lungs. They seem to be less troubled with symptoms and too often their cough is attributed to the evils of age and emphysema. Alexander B—, æt 50 years, is a good case in point. He had been troubled with cough for three years but did not seek relief because it was so slight. He was asked to become an out-patient, but his general condition and appetite were so good that after a few visits he ceased to come any more. Those advanced in years do really seem to be

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little distressed by the disease and carry on their vocations with but slight discomfort. Nevertheless this patient had involvement of three lobes of the right lung. His wife had had thirteen children, ten were dead, six of them had had tuberculosis of the lungs, one died of meningitis, three died quite young of wasting disease; and you will not wonder that when one of the surviving children was brought up as an out-patient for cough it was considered necessary to examine the parents; the father was found to be in the condition above mentioned. In advancing years the disease progresses insidiously, with all the greater risk to the family of the afflicted one. No doubt the aged bear tuberculosis well because they are examples of the survival of the fittest, but such survivals ought to be properly labelled and warned of their danger to others.

Habits.—It is uttering a platitude to say that chronic alcoholism is an element of unfavourable prognosis; you know only too well how grave are the cases of alcoholic neuritis who are also the subjects of tuberculosis of the lungs. It is a favourable opportunity to remind you of another out-patient at this hospital. He came for increasing weakness and very slight cough: he was examined repeatedly with negative results, and in despair he was sent to University College Hospital as a case of possible cirrhosis of the liver, as he was a chronic alcoholic. Fever was present and his cough suggested the examination of his sputum, which was mucoid and suggested only bronchitis; tubercle bacilli were found, but it was only some weeks later that signs were detected in the lung. He had marked emphysema, and no doubt this disguised his pulmonary tuberculosis. Some writers even say that emphysema in a tuberculous patient is an element of favourable prognosis; quite possibly this is so, but it is also a serious complication from a diagnostic point of view, for emphysema obscures the more serious tuberculous disease. Occupation within doors and working in atmospheres loaded with dust and other such factors should be considered; they must be altered if your patients are to be helped to make their disease pursue a slow course—an object which should always be aimed at, whether we believe in the absolute curability of this disease or not: a boy employed in underground rooms by a firm of fashionable photographers has done much better since taking up the work of a van-boy.

(2) *The character of the disease.*—Next to the general aspect of your patient and his habits, etc., pay close attention to the behaviour of the disease itself; nothing is more valuable in forming a prognosis than the careful study of the antecedent course of the disease. You know the old German story of the pedestrian who asked a peasant how long it would take him to reach a certain village. The answer to "go ahead" was not deemed satisfactory and an explanation was asked for such odd advice. The reply was simple, viz. that it was impossible to say how long it would take the traveller until the peasant knew how fast the traveller could walk, and he merely wanted to observe that. So with pulmonary tuberculosis; it is impossible to attempt a prognosis with the means ordinarily at our disposal unless we know the antecedent course of the malady. For the purpose of this lecture I have analysed 275 cases of pulmonary tuberculosis amongst the out-patients attending this hospital; thanks to the energy of several clinical assistants, these cases have been carefully studied. A record has been made in each of these cases of the occurrence of any malady which is conceded to be a proof that the patient has become the host for the parasite of this disease. Each of these cases has tubercle bacilli in the sputum. In some the signs are almost beyond dispute; in others they are so slight or so different to the usually accepted signs that several of your number, the clinical assistants who have helped me, and myself have been misled and have considered the physical signs of tuberculosis as doubtful or in some cases absent altogether. The disorders, etc., which have been considered as being possibly the earliest clinical expression of tuberculosis are such as the following:

Pleurisy, with or without effusion	59 cases.
Hæmoptysis, slight or marked	25 „
Inflammation of the lungs, pneumonia, or congestion of the lungs	21 „
Dyspepsia	5 „
Fistula in ano	2 „
Abscess of the ribs	1 „
Glands in the neck	4 „

Other manifestations which were possibly earliest signs were:

Bronchitis	132 cases.
Influenza	13 „

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The remaining 13 cases gave a history of previous illnesses which can hardly be correlated with tuberculosis, such as St. Vitus'dance, acute rheumatism, debility, colds, etc.

It is interesting to turn now to the various figures which have been given as an indication of duration of life when tuberculosis *has been diagnosed.* You are all probably familiar with them in the description given by Dr. Kidd.

Louis: Death occurs in less than 9 months in more than half the cases.

Louis and Bayle: Average duration 1 year 11 months.

Dr. C. J. B. Williams and Sir J. Clark: 4 years.

Dr. C. Theodore Williams: (198 cases) $7\frac{3}{4}$ years as observed in private patients to a fatal termination.

Dr. J. E. Pollock: 2 years $6\frac{3}{8}$ months for 3500 cases whilst under observation as out-patients at this hospital; taking into consideration the whole duration of the disease, the average life is over 4 years.

Dr. Kidd from his experience during twelve years' work amongst the out-patients of this hospital thinks the four years average nearer the mark than the two years.

During the last three years, over 4000 cases have presented themselves for treatment, but those of you who work here know that these are not all cases of pulmonary tuberculosis; nor does the number 275 represent all the cases of pulmonary tuberculosis; it represents the number of cases taken without any special selection, early and advanced, in whom tubercle bacilli have been established in the sputum—a surer test than the ordinary physical signs for all cases of hæmoptysis, of marked apical contraction, and even of apical cavities with purulent expectoration are not necessarily cases of tuberculosis though it may be argued they were at one time. I can offer no answer to this last objection beyond saying that the probabilities are that tubercle bacilli are not the cause in all of them, and we have had an example of apical bronchiectasis in which post-mortem none of the ordinary microscopic signs of tuberculosis, such as giant cells, etc., were demonstrable.

I have made a table of the duration of symptoms of the disease at the time when the patients presented themselves for examination, and were found to have tubercle bacilli present in the sputum; the extent of involvement is also given.

Duration of symptoms on first examination.	No. of cases.	No. of lobes involved on first examination.				
		1	2	3	4	5
3 months ...	40	1	14	8	13	4
6 „ ...	34	0	9	7	8	10
9 „ ...	22	0	8	7	3	4
1 year	23	1	3	7	6	6
2 years	41	0	6	6	17	12
3 „	16	0	5	4	5	2
4 „	23	0	6	3	6	8
5 „	13	0	2	5	2	4
6 „	9	0	2	2	2	3
7 „	3	1	0	2	0	0
8 „	4	0	0	1	1	2
9 „	3	0	2	0	1	0
10 „	7	1	1	2	0	3
11 years	2	0	2	0	0	0
12 „	3	0	0	1	1	1
13 „	2	1	0	1	0	0
14 „	2	0	2	0	0	0
15 „	3	0	0	0	2	1
16 „	1	0	0	0	0	1
17 „	2	0	0	0	1	1
18 „	4	0	1	1	0	2
19 „	0	0	0	0	0	0
20 „	1	0	0	0	0	1
20–30 years ...	6	1	2	0	1	2
30–40 „ ...	3	0	0	1	2	0
Period not defined	8	3	2	0	1	2

This list of 275 cases includes a number of patients who cannot be said to be cases in which the lungs were the first organs to be attacked. Various other organs and tissues were involved, such as the rib, cervical glands, etc. The number of cases in which lungs were presumably affected, including those in whom bronchitis and cough, pleurisy, hæmoptysis, inflammation of the lungs were the first evidence of possible tuberculosis, amounts to 237 cases, or about 88 per cent.—*i. e.* about 33 of the total 275 cases may be objected to as not being tuberculosis of the lungs in the first instance. Let us exclude 33 cases of the longest duration, and then even you will see that there are 230 odd cases in whom the disease had been present from three months to twelve years. [It is stated by

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Aufrecht that chronic tuberculosis of the lungs often lasts ten years.] If the above figures are accepted, then we are brought face to face with the knowledge that the average period during which lung symptoms were present in the 237 cases of tuberculosis of the lungs was two and a half years when the patients first sought relief and were examined. It will be more instructive to remember that about one fifth to one sixth (43 out of 237) of the cases had had symptoms for longer than the average duration of life as given by Dr. J. E. Pollock, viz. four years, and were able even to be up and about and attend as out-patients. One out of every five or six patients suffering from tuberculosis, and presenting themselves for the first time for medical examination, will by simple measures, drugs, diet, and hygienic precautions live longer than four years. One case I will describe in a little more detail. Mrs. A. G—, æt. 58 years, came to this hospital for the first time over twenty-four years ago, and was treated by Dr. Burney Yeo and Dr. Kingston Fowler for frequent hæmoptysis, cough, emaciation, and pallor. The hæmoptysis persisted and six years later no physical signs could be discovered. She appeared again in January of this year, having been troubled with hæmoptysis, copious yellow phlegm, and diarrhœa. She was thin and pale, had a temperature of 99·6° and a pulse of 100. There were suspicious signs at the right apex involving only one lobe, and tubercle bacilli were present in the sputum.

This case will serve to show how protracted may be the course of pulmonary tuberculosis, for it can hardly be doubted that her hæmoptysis, which was such an early and persistent feature, was due over twenty-four years ago to tuberculosis.

This short study of the course of the disease will show that it is a very important matter to consider the chronicity of the symptoms before your opinion is sought; the longer the symptoms have existed the longer, within limits, will the patient live.

Then as for special symptoms, increased and maintained pulse frequency (over 120), elevated temperature, failing appetite, progressive loss of weight, copious expectoration, marked dyspnœa, diarrhœa, fistula in ano, tuberculosis of the genitalia, and highly offensive odour of the breath are signs of bad omen.

Whenever a prognosis is being made reserve should be made for the possibility of such grave complications as gangrene of the lung, pneumothorax, meningitis, empyema, tuberculous peritonitis, spinal caries, tabes mesenterica, lardaceous disease, diabetes mellitus, and for dropsy occurring in one or both legs. Such developments as these are often heralds of impending death in the near future. If pneumonia occurs and complicates tuberculous disease in young people between sixteen and thirty years, death, if not rapid, occurs generally within one to three years (Aufrecht).

Early hæmoptysis is not a very serious symptom, as shown in the case of Mrs. A. G—, but if the bleeding is marked and persistent the outlook is grave: curiously death from hæmoptysis is very rare. A word or two is necessary about the occurrence of tuberculous laryngitis; if diffuse and infiltrating it is a grave complication. From the practical diagnostic point of view it is most important to remember that this complication obscures the amount of disease in the lungs, and when estimating the degree of severity and the extent of lung involved in such cases it is imperative to remember that the signs of disease as found by physical examination, and which are notoriously, as elicited by the most skilled observers, imperfect guides to the true state of affairs, are still more difficult to obtain in laryngeal cases.

Muscular irritability, myxœdema, and intensification of the nuchal hollows are signs of ill omen.

Amongst the favourable signs other than the absence of the above conditions are marked fibrosis and calcification; pneumoliths are coughed up occasionally in cases in which the latter process is proceeding satisfactorily. It is said that cases beginning with pleurisy are more favourable than those in which the lung itself appears to be the initial seat of disease.

(3) *Skill in examination of the patient.*—This is the third subject of great importance in the prognosis of the disease. It is impossible in this lecture to detail the methods of examination which are necessary to exclude or to diagnose tuberculosis of the lungs, but attention to such simple matters as the position of the patient, examination being carried out both in the sitting and recumbent position, is essential. The common seats of disease, such as the apices of the lobes and the lines of the interlobular septa, and especially the dome of the axillæ, should be thoroughly explored. When in doubt ask your patient to cough in order to bring out fine crepitations, and when your physical examination is negative examine the sputum in cases of suspected open tuberculosis. In closed tuberculosis frequent weighing, the tuberculin reaction, or the estimation of the opsonic index by Professor Wright's method, are essential.

Ehrlich's diazo-reaction is used as a prognostic test and Holmgren finds it present almost constantly in Turban's advanced second and third stages. When the reaction is intense the maximum duration of life is six months, the average duration two months. Moderate reaction means on an average six months' life, but life may be continued for one and half years.

June 25th, 1906.

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SOME SURGICAL RESULTS OF IMPROPER FEEDING.

A Post-Graduate Lecture delivered at the Hospital for Sick Children.

By EDRED M. CORNER, M.A., M.B., B.C.Cantab., B.Sc.Lond., F.R.C.S.Eng.

Senior Assistant Surgeon, Hospital for Sick Children, Great Ormond Street; Surgeon to Out-patients, St. Thomas's Hospital.

LADIES AND GENTLEMEN,—I would remind you of the old fable in which the various organs of the body quarrelled with the stomach because it appeared to be idle. They then discovered that without the stomach they quickly became weak and useless. Digestion and absorption, *i. e.* nutrition, are the most important functions which a child must perform. The fable also teaches us that all parts of the body are affected by and dependent on the performance of the functions of the alimentary tract, represented by the stomach in Æsop's tale. So that good nutrition will affect all parts of the body for good and bad nutrition will affect all parts for evil. Hence, although we may pay scant attention to good nourishment and good growth, we should pay very much attention to improper feeding on account of its ill effects being both deleterious and universal throughout the body. Further, childhood, in virtue of its property of growth, is the most important epoch in life to consider; for as bad feeding leads to bad growth, so may the seeds of future disabilities be laid down. It is not practicable to consider within this lecture all the possible results of improper feeding, so that only those which come under the notice of the surgeon will be mentioned, and not all these. Stress will not be laid upon such diseases as are well known to result from improper feeding, as rickets, scurvy, or intussusception; but attention will be directed to surgical conditions which are not generally recognised as being so caused.

The results of improper feeding may be due to faults on the part of the child, as when suffering from some ailment; the food, as when it is unsuitable; or, and most often, both the food and the child are at fault. The practical lesson to be learned is, that although a child may be supplied with the best food, it may be wholly unable to digest it and so suffer from the consequent ills. Many and many a time symptoms have been found to occur in breast-fed children. It should be remembered that mothers' milk is not a panacea for all infantile ills.

At birth the meconium in an infant's bowels is usually sterile. After birth organisms quickly obtain admission and multiply in the intestines. The babe is unaccustomed to the action of their chemical products, and should the rate of inception of the organisms be too great the baby will suffer from intestinal disturbances. These organisms may have a very profound effect on the child and its feeding. Suppose a baby to be fed with its natural food, its mother's milk, it will be able to digest and utilise a certain amount. Should there be any *excess* of this amount, the organisms in the bowel will cause its decomposition, just as they would do if outside the body. For our purpose the fermentation of proteids, carbohydrates, and fats in the intestine may be compared with the fermentation of sugar by yeast. When sugar undergoes fermentation, two important substances are formed, amongst various by-products, alcohol and carbonic acid gas—that is to say, a toxine and a gas. When proteids, carbohydrates, or fats ferment, in a similar way, toxines and gases are found amongst the many products. Any residuum of nourishment, even if it is of the very best, will go bad and ferment. If it is quickly defæcated, all is well, but if it is retained and the condition remains for some time, then the child's health will suffer. Hence it is the overfed rather than the underfed baby which develops rickets. The children of the well-to-do classes are more abundantly fed than are the children of the poor; hence the prevalence of rickets and other ailments amongst the former. Similarly, rickety babies are frequently fat and not lean, the result of the plenteous supply of food. Seeing that proper as well as improper food becomes poisonous when it decomposes, great care should be given to the bowels and to the character of the motions of a young child, as they will indicate the conditions present.

There are three lines to be followed in considering the consequences of food fermentation within the bowel, namely the effects of the toxines, the effects of the gases, and lastly, the effect of the fermentation of the bowel itself.

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(1) The Effect of the Toxines.

The poisonous products, the toxines, are absorbed from the intestine and carried by the portal vein to the liver, which is the first organ, apart from the alimentary tract, to be affected. As you know, the liver usually becomes enlarged under such circumstances. But its enlargement takes time, and the general system during this period is being poisoned both indirectly, through the hepatic veins, and directly, through the intestinal lacteals, the receptaculum chyli, and the thoracic duct. It will be obvious that this is the stage when the child's condition will be most advantageously diagnosed, treatment being then easier and more sure. This stage can be recognised readily, the observer being more likely to be misled by its great frequency than by anything else. The symptoms are: the child is querulous, often crying and not taking its food so well; it does not sleep well; its pillow is often wet from perspiring about the head; its bowels are inactive or loose; its abdomen is large and may show multiple herniæ, a protrusion between the divaricated recti, and bulging flanks; it suffers from wind and the stools are offensive. To put it generally, the child is obviously ailing and uneven in its methods of life and in the performance of its various functions. Sickness indicates gastric irritation, and therefore improper food rather than excess of good and proper food. Hence it need not be present at all. In spite of its improper feeding the child is often fat and apparently well cared for.

At this stage the treatment is easy. The diet must be attended to both with regard to quantity and quality; the bowels must be regularly evacuated, and the child's stools and general condition watched to see the results. As the fermentations are acid fermentations, it is good practice to combine the purgative with an alkali, as in a powder of rhubarb and bicarbonate of soda. If it is recollected that when a child swallows a foreign body several days may elapse before it is passed *per anum*, it will be realised what a great deal of decomposition can take place in food residues before they are got rid of.

Too much stress cannot be laid on the importance of the recognition and treatment of this initial phase of intestinal intoxication, or, as it might be termed if its ultimate results are borne in mind, the pre-osseous phase in rickets.

If this phase is allowed to pass, then the toxines will exert their actions on all the tissues in the body. When the bones are affected, and have been so for some considerable time, the condition is recognised by the enlarged epiphyses etc., characteristics of rickets. When the vascular system is affected scurvy or anæmia appear. Should both systems be affected simultaneously, the disease is called scurvy-rickets. But these diseases only represent the affection of two systems! We do not know what may be happening in the other parts of the body. If we were to guess from what we know occurs in the bony and vascular systems, we must infer that gross and grave changes are going on elsewhere. It is dangerous to hazard a guess as to what diseases or deficiencies of organs are thus brought about. But it is salutary to think that as great harm may be done in brain and nervous system as in the bones.

(2) The Effects of the Gases produced by Fermentation.

As the meconium in the bowels of a newly-born infant is usually sterile, no decomposition should have occurred and no gas should be present. When organisms obtain admission to the alimentary canal, fermentation will begin and gases will be produced. A certain amount of fermentation is essential and seems physiological to the adult organism, childhood being utilised as the time for inuring the organism to the ordinary intestinal poisons. One result of the gas-formation is to increase the intra-abdominal tension, the effect of which will be felt mostly by the weaker parts of the abdominal wall. These weak parts are the linea alba, the umbilicus, and the inguinal regions. If the gas-formation is large, hernial protrusions may appear in all these parts. With regard to the inguinal region, it matters not if a vaginal or funicular sac is already present: it is the increased intra-abdominal tension which forces the bowel or omentum into the sac. Otherwise the tension has to accomplish the additional task of forcing a peritoneal sac down in front of the hernia; this would be an acquired hernia sac.

It is not unusual that when a congenital abnormality occurs it is accompanied by other congenital abnormalities. But when two inguinal herniæ, an umbilical and a protrusion between the divaricated recti are present, it is rather more logical to

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associate their presence with one common cause. Especially is this so when there is also a large abdomen with bulging flanks. There can be no doubt that many of the herniæ of children arise in this way, but as it is impossible to distinguish morphologically between the funicular and the acquired sac, it is impossible to say how many. Nevertheless, it is a fact that simple non-operative treatment will cure many of these herniæ and relieve a number more, obviating the immediate need for operation.

If these herniæ or the abdominal distension alone are present for a long time, they or it will lead to permanent weaknesses on account of the fact that the normal healthy growth which should take place in the abdominal wall has been modified by their presence. Hence, if they are allowed to persist *they may sow the seeds for the development of a hernia in the adult*, although they may not actually produce one in the child. The relief of the distension and the cure of the herniæ whilst the child is still young are very important for the future welfare of the adult.

There is still another important effect, besides that on the hernial rings, which gas-formation and prolonged distension of the abdomen can produce, namely the effect on the abdominal wall. The back of the abdomen is well supported by the spine and its muscles; the lateral part of the abdomen is also well supported by the ribs, ilia, and muscles; the front of the abdomen is only supported by long muscles, and in consequence bears the brunt of the abdominal distension. The recti are strips of muscle with bony attachments at either end, and as such suffer comparatively little. The portions of the external and internal obliques, particularly the latter, which come into consideration here, being flat, broad muscles, are stretched by the abdominal distension both lengthways and broadwise, causing the separation of their component muscular bundles. Now, if you stretch an elastic sheet in both its superficial directions, when the stretching forces are removed the sheet returns approximately to its former size; but if, instead of a passive elastic sheet, an actively growing muscle is stretched for some time, on account of the multiplication and growth of its fibres, that muscle will not return to its original length when the stretching forces are removed, having by its growth adapted itself to the increased length. Hence, after a child's abdomen has been distended for some time the external and internal obliques, the latter muscle suffering the more, are permanently stretched and redundant. This is evidenced by the bulging flanks so often seen in children. In this way another source of weakness may be transmitted from the child to the adult, rendering the latter disposed to hernia-formation, should such not already have occurred.

(3) The Effects of Fermentation on the Intestine.

The decomposition in the intestine leads to numerous changes in it. Gastritis indicates that improper food rather than excess of good food

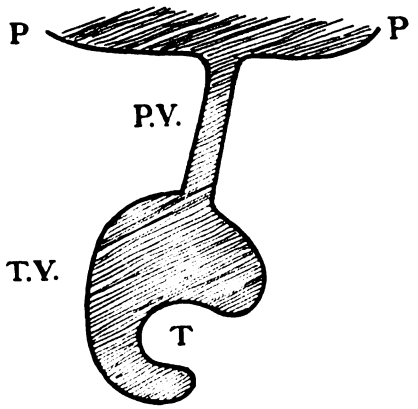

Fig. 1.—Common hydrocele of children, with patent processus vaginalis communicating with the peritoneal cavity and the tunica vaginalis. P.P., the peritoneum; P.V., the processus vaginalis; T.V., the tunica vaginalis; T., the testis; the shaded portions represent the fluid of the hydrocele. A hydrocele in a hernia sac.

is responsible for this condition. Enteritis and colitis show themselves by pain, diarrhœa, mucus, and perhaps blood, in the stools. A secondary infection with tubercle bacilli may lead to tabes mesentericæ. Intussusceptions and prolapsus recti may occur. As the child has abundance of lymphoid tissue about its cæcum and appendix, it usually escapes appendicitis, such as would occur in an adult. The above are all changes within the bowel, and are accompanied by some subacute inflammation of the coats of the bowel. The inflammation of the mucous coat shows itself in diarrhœa, mucus, and blood in the stools. Inflammation of the muscular coat leads to inaction of the bowel and constipation. Inflammation of the peritoneum over the bowel leads to the presence

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of a small quantity of fluid in the peritoneal cavity, and should a patent processus vaginalis be present, to the formation of a hydrocele of the tunica vaginalis.

The Origin of some Cases of Varicocele.

We have now finished our brief sketch of the surgical results of improper feeding. There are one or two problems still to consider. The first is that of varicocele. It is not customary to look for the origin of varicocele in improper feeding. It is a surprising fact that right-sided inguinal hernia form about 60 per cent. of all the herniæ of children. The presence of a hernia in the inguinal canal, unless it is tightly prolapsed and nearly strangulated, will remove any obstruction to the return of the blood in the veins of the right pampiniform plexus. Therefore there is reason that the right side should not be affected with varicocele. On the other hand, we know that the left inguinal region does not give way nearly so frequently, leading to the formation of the hernia. So it will be of interest to see what will be the result of this not giving way. The inguinal canal has a valve-like action, as seen in Fig. 2. If a hernia forms, the valve is destroyed; if a hernia does not form, when the abdomen is distended the valve is not destroyed but closed tighter than before, obstructing in consequence the venous flow in the pampiniform plexus. We see, therefore, that there is reason to expect the right side to be free from varicocele, whilst there is reason to expect it on the left. If care is taken at operations on children to observe the veins, especially those of the left pampiniform plexus, it is not uncommon to find miniature varicoceles in quite young children. Such observations support the above view, that the *seeds of varicocele are often sown in childhood.*

Another great cause for the predominance of left-sided varicocele is that the left pampiniform plexus has two venous supplies; namely, from the left spermatic vein which joins the left renal vein, and a vein, or venæ comites, which accompanies the spermatic artery and opens into the inferior vena cava. The right pampiniform plexus has only one connection.

The varicose veins of the pampiniform plexus of the child remain unnoticed until puberty, when the testis takes on rapid growth and functional activity. Then the condition of the veins, on account of the increased work which they have to do, becomes obvious and is called a varicocele.

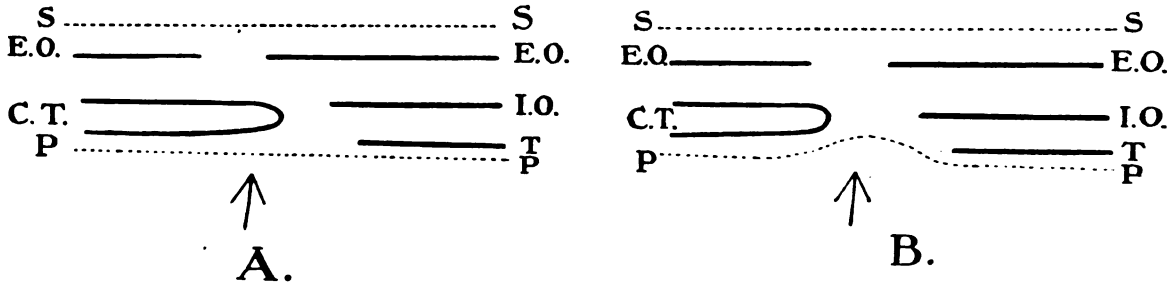

Fig. 2.—A. Diagram of section of the healthy inguinal canal. Note that the edge of the conjoined tendon (C.T.) overlaps the external pillar of the external abdominal ring. A valvular mechanism is thus established. B. Similar section, but in this the internal abdominal ring has become larger and the valvular mechanism seen in A is abolished.
The arrow indicates the direction of the intra-abdominal pressure; E.O., external oblique muscle; I.O., internal oblique; T., the transversalis; C.T., the conjoined tendon; S.S., skin; P.P., the peritoneum.

There can be little doubt that the presence of varicoceles in young adults can often be explained on this hypothesis, though it would be foolish to attempt to explain all varicoceles in this way. There is a further stage in the development of a varicocele to which attention may be turned. If the venous obstruction is chronic and attains sufficient degree, it will set up a *chronic inflammatory condition of the tunica vaginalis, leading to the formation of a hydrocele.* In this way the occurrence of hydroceles in young adults receives explanation, as does also the fact that they recur and recur after simple tapping. The production of hydroceles, spermatoceles, and other changes as the result of venous obstruction is well illustrated in the paper written by Mr. Nitch and myself in the 'British Medical Journal,' January 27th, 1906, on the results of operative treatment.

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From the above sections *it will be seen that the seeds for the formation of herniæ, varicoceles, and hydroceles in adults can be sown in childhood*—an example of the fact that the child is parent to the adult even with regard to the ills developed by the latter.

The Naso-pharynx and Adenoids.

If one thinks over the rickety affections of the skull which are known, it becomes obvious that we recognise gross changes as occurring in the vault of the cranium, whilst no attempt has been made to ascertain if any changes are to be found in the base of the skull. Of course, the vault is developed in membrane and the base is laid down in cartilage. But we know that bones which develop in cartilage suffer from rickets, as can be seen in other parts of the skeleton. So there is every reason to expect that bony deformations will be present in the base of the skull just as they are in the vault. The base of the skull is an awkward situation to study on account of the difficulty of making observations. The bones, as elsewhere, must become thickened and, later, sclerosed. What will be the effect of this? The naso-pharynx will become encroached upon and rendered smaller, making it difficult, or at least uncomfortable, to breathe through the nose. If the obstruction is sufficient to cause a negative pressure in the nose, the surrounding softened membrane bones, such as the nasal and palate bones, will fall in. The bridge of the nose will sink and the palate become high, surely converting the child into a mouth-breather. The unventilated naso-pharynx forms a kind of *cul de sac*, becoming the breeding growth of the organisms which form the flora of the nose and mouth, such as the appendix does for the large intestine. The irritation afforded in this way leads to the growth, usually called the overgrowth, of the lymphoid tissue present, giving rise to the condition we recognise as adenoids. The naso-pharynx is now stuffed up with adenoids and the bones have enlarged and fallen in, stopping nasal respiration. If a limb is functionless, it will grow imperfectly; similarly the naso-pharynx. That cavity, having been rendered functionless by the absence of nasal respiration, grows much less than it would do if it had been functional. The thinness of the nose, the poor development of the upper jaws, etc., seen in the faces of adults with neglected adenoids, is usually attributed to this. But there is another point to which I would direct attention, namely the influence of rickety changes in the bones and of the absence of nasal respiration upon the development of the air-cavities in connection with the nose, such as the sphenoidal sinus. The imperfect development of these air-containing spaces will aid materially in stamping upon the face of the adult the stigma of neglected adenoids, the hall mark of imperfect growth and function.

From the above it will be seen that there is a very intimate connection between improper feeding, rickets, and adenoids. Further, the relationship illustrates that the proper performance of its functions alone leads to the proper growth of the part, whilst bad physiology of a region will lead to the bad anatomical development of the part—a lesson which should be always kept in mind when treating deformities of the body, function, or mind in children.

If one remembers the stage of intestinal toxæmia before rickety changes have already occurred in the bones, it will always be realised how often cases sent to hospital as adenoids merely want dieting and treating, followed by a course of nasal respiratory exercises, and do not require operation, and if they are operated on, the result is imperfect or a complete failure. Knowing that obviously rickety bones become perfectly straight as the result of treatment and healthy growth, the proper treatment must be adopted and time allowed to elapse for the rickety changes in the bones of the naso-pharynx to be removed before a final prognosis can be given. Certain it is that many of these children avoid the need of all operative treatment; whilst if the medical treatment is not adopted and they come at last to surgery for aid; when owing to the previous neglect, it is often impossible to do much good.

The Relation of Congenital Syphilis to Rickets.

There can be no doubt that there is a very intimate relationship between these two diseases. An authority* writes that "congenital syphilis is often a disease in which general malnutrition is the most prominent feature." There is no state-

* Mr. Jonathan Hutchinson, Jnr.

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ment more truthful than this, and in it lies the kernel of the confusion between these two diseases. It is the "malnutrition" which produces the rickets. The syphilitic child may not be able to digest and absorb the very best of foods which can be given to it. Further, if the accounts of the two diseases are read side by side, it will be seen that there is much in common. For instance, the skulls are similarly malformed, they develop similar nodules, and the long bones are similarly affected. In both diseases the vault of the skull shows the condition of *craniotabes*. The characteristic feature of ossification in rickets is its irregularity. In a bone developing in firm cartilage this irregularity will not be very noticeable to the touch; but should the bone be developed in softer membrane, it must present hard and soft areas as they are ossified or not, such as is described under the name "craniotabes." It is now obvious why that condition has been noticed in the thinner portions of the cranial vault, which are developed in membrane.

The same authority quoted above says also: "It will be noted that considerable resemblance exists between the bone lesions due to syphilis and those due to rickets; the latter, however, occur usually at a later period." The two diseases, certainly, are confused. In fact, it can be stated that every syphilitic child must suffer in some degree from improper feeding, frequently to the extent of developing rickets, but it is only a very small percentage of rickety children who are syphilitic. To diagnose infantile syphilis it is necessary to recognise definite syphilitic signs and stigmata, such as *cannot* be attributed to rickets. There can be no doubt that congenital syphilis is a maligned disease, having much attributed to its specific action which is due in reality to the rickety conditions which it may produce.

Improper feeding in the adult produces its results just as it does in the child, the great difference being that one has ceased to grow whilst the other has not. The absorbed *toxines* cause changes in the liver—*e. g.* cirrhosis and general fatness, obesity. The *gas-formation* causes the distended protuberant abdomen. So that the fat, pot-bellied adult is the matured type of the deformed rickety child.

Summary.

There is no more important function for its welfare performed by a child than that of nutrition. The foregoing lecture has brought forward so many practical points that it has been thought best to epitomise them.

(1) The best foods, unwisely administered, can produce symptoms of improper feeding. They can often be seen in breast-fed babies.

(2) The fault may be with the child and not with the food.

(3) At birth the alimentary canal is sterile. With the admission of organisms begins the decomposition of food residues and the "intestinal education" of the child.

(4) There is a recognisable stage of toxæmia before any rickety signs are manifest. Treatment should be adopted then.

(5) The *toxines* are known to produce gross changes in some organs—rickets, scurvy, etc.; presumably, therefore, they do so also in other structures as yet unknown to us.

(6) The *gas-formation* may produce inguinal and umbilical herniæ, divarication of the recti, large abdomen, bulging flanks, hydroceles, etc., also sowing seeds of weakness for the possible future development of herniæ, varicoceles, hydroceles, spermatoceles, etc., in the adult.

(7) In the *intestine itself* many changes may occur—gastritis, enteritis, appendicitis, colitis, intussusception, etc.

(8) Adenoids are commonly a sign of rickets or improper feeding.

(9) Congenital syphilis is held responsible for many rickety changes.

(10) Childhood is the period in which the individual is rendered immune to the ordinarily formed intestinal toxines, which become in consequence harmless to the adult.

June 25th, 1906.

In view of the approaching elections of Direct Representatives it is most important that all practitioners should assure themselves that they have their correct address inserted in the 'Medical Register,' for otherwise not only will they possibly lose their votes, but they will also run a considerable risk of having their names erased from the Register because we are unable to get into communication with them. No circulars of inquiry have been issued this year, as the sending out of voting papers will in itself form a means of correction.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

A CASE OF CARDIAC DISEASE.

* Delivered at St. Bartholomew's Hospital.

By W. P. HERRINGHAM, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN, — About a year ago a man was admitted under my care in Colston Ward with symptoms of cardiac disease. He was about 43 years of age, and he said that, beyond having had chorea when he was a child and pleurisy at some unknown period of his life, he had had no illness until during the last five years. During that five years he had had three separate attacks of rheumatic fever. That was all the history of previous illness which we could get. For three months before coming in he had noticed dyspnœa, and for two months he had had swelling of his legs, which gradually spread upwards into the abdomen. When he was admitted we found that he had considerable œdema of the legs and of the loins, that there was a good deal of fluid in the abdomen, that his liver was right down to the level of the umbilicus in the nipple line, therefore very much swollen, and that pulsation was being conveyed through it. In addition, he was markedly cyanotic. Of course those two symptoms, cyanosis and dropsy, at once turned our attention to the lungs and the heart. But on examination of his lungs it was fairly clear that there was no explanation of his condition to be found in them. All I could find was that he had a small patch of dulness at the right base, which might have been either thickened pleura or a little pleural effusion, or a little congestion in that base, or even nothing but collapse due to the ascites or the enlarged liver. So I turned to his heart, and in it I certainly found definite signs of disease. His apex beat was one inch outside the nipple line,

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and there was a systolic and later a presystolic murmur, which made me certain he had mitral stenosis and regurgitation. But I have often told you, and I cannot repeat too often, that the exact number of murmurs that a patient has, or the exact condition of his valves, is not an explanation of his disease, nor do they help very much in the matter of prognosis. There are plenty of people going about the world with double mitral disease, and doing hard work too, and some with double aortic disease. But when such patients fail the explanation is not to be sought in their valves, but in the condition of the heart wall itself. They break up because the muscle goes. The valves are not in any degree worse than they were a year or two or three years before.

So, of course, in the present case the examination was conducted from the point of view of failure of the myocardium. And such a condition can be easily recognised. It betrays itself partly by anatomical and partly by functional alterations. The anatomical alterations consist of dilatation, and the functional consist of palpitation of the heart and irregularity of the pulse, both in regard to force and rhythm, with lowered blood-pressure. Let me stop for a moment to impress upon you the necessity of conducting your examination to these parts with the greatest accuracy. With regard to the dilatation and the area over which that dilatation extends, I am always sorry when I see people content with finding the superficial area of cardiac dulness. Although that is of some use, it is even more dependent upon the condition of the lungs than upon the condition of the heart, and it bears no comparison in importance with the actual outline of the heart, which can be traced if you practise yourself in the detection of deep dulness. In 90 per cent. of the cases the deep dulness is an easy thing to make out. I showed many years ago, and other people showed also, by experiments on the dead body, that you can accurately map out the outline of the heart within less than a quarter of an inch all the way round. That is a piece of knowledge of infinitely more importance than mere percussion of superficial cardiac dulness. In the same way I may mention the examination of pressure in the blood-vessels. That is one of the most important functions in the body, and I am always sorry when I see people trying to do it with one finger or with two fingers of one hand. You cannot do it so: you must practise yourselves in using one finger of each hand, one to stop the pulse and the other to tell when it is stopped. If you try two fingers or three fingers, with one pressing down in the middle, and only use one hand, you cannot distinguish between the three fingers. It is a very useful practice, when you can, to correct that sort of estimate by using an instrument such as I show you here. It is a Riva Rocci sphygmomanometer. We tie this bag round the branchial artery and blow it up, so that the pressure which it takes to stop the brachial or the radial pulse is visible in the mercury manometer. But this does not take the place of the finger, because the instrument can only measure the systolic pressure. It takes no account of the lesser blood-pressure of diastole, which the finger can accurately recognise. If you think of what happens in aortic regurgitation you will understand that an enormously hypertrophied ventricle will pump so great a volume of blood into the arteries that the pressure will materially rise for the moment, but the regurgitation will immediately allow it to sink again, as can be recognised with the finger. So the sphygmomanometer may register high, and yet the mean blood-pressure, which is the most important, may be much below the normal. When we came to examine this man's heart we found no evidence of much dilatation. The apex beat was outside the nipple line, it is true, but so is every apex beat where the heart is a little hypertrophied, and the heart in nearly every case of mitral regurgitation extends that much. The most important part of the heart, if you want to examine for dilatation, is the right border, the area occupied by the right auricle, because it is not until that is distended that there is evidence that the backward pressure has forced the safety-locks of the heart, and has come to impinge on the circulation in the veins, and dammed back the current in them. The right border of this man's heart—that is to say, the border of his right auricle—when examined by deep percussion, did not extend more than an inch beyond the right edge of the sternum, and that is very little more than natural; it is usually half to three quarters of an inch from the right edge of the sternum. Again, his pulse was regular, there was no palpitation about the heart, the rate of which was 100 to the minute, and the blood-pressure was natural to a man of his age. So neither in the

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anatomical relations nor in the functional condition of the heart could we find valid evidence that he had much myocardial failure. Under those circumstances we were puzzled to account for the extreme venous congestion which was betrayed in his cyanosis and ascites, his swollen legs and back. At last I concluded that the condition in the heart itself could not account for it, and that there must be something further interfering with the venous circulation. I ultimately said that I believed that outside the heart, and probably connected with the rheumatic affection which we knew he must have, there was an extension of inflammation which had involved the tissues surrounding the heart, and had pressed upon the great veins. And, in fact, that is what was found to be the case, for the man eventually died, and we found post mortem that he had, as we knew, mitral stenosis and regurgitation; secondly, that he had, as we suspected but could not prove, adherent pericardium; thirdly, that he had spreading from his pericardium an immense increase of fibrous tissue in the mediastinum, which had constricted the vena cava and fixed the heart to the surrounding tissues. That fully explained the fact that there was much greater obstruction to the venous circulation than was justified by the intrinsic condition of the heart. And we found also that there were adhesions in the pleura and chronic peritonitis with adhesions to various parts of the abdominal cavity. That was one case.

Another case, rather different from that, came under my care the other day. I was called to see the case by Mr. Lockwood in Kenton Ward. The man was 39 years of age and of temperate habits. A year ago he had pleural effusion on the left side, for which he had been tapped two or three times in other hospitals. Since that time he had lost two stone in weight, and for the last three months he had had a cough, and some amount of sputum had been brought up. He was, like the first man, markedly cyanosed about the head and neck, obviously a great deal wasted; there was a slight amount of impairment on percussion at the right apex, and there was râles there. There was some dulness at both bases, but there were breath-sounds audible over both and a certain amount of vocal vibration. Further than that, his abdomen was very prominent. The surgical notes previously made recorded that his liver had been two inches below the ribs. That was never the case in the medical wards. But the abdomen was very prominent, transversely creased at the umbilicus, as it is often when there is ascites, and resistant as in the tubercular peritonitis of children. Putting these things together as well as I could, I concluded that in him too there was chronic peritonitis. I thought from the appearance of the umbilicus there was probably some amount of fluid in the abdomen, though there was no movable dulness. I thought the dulness on the two sides of his chest was probably accounted for by the old pleurisy on his left side which we knew he had and by recent pleurisy on the right. The heart was quite natural as far as we could tell. There was a further symptom in the chest, namely that all over the precordial area there was visible retraction of the upper spaces with systole of the left ventricle. As the apex beat came forward the chest was considerably retracted all over the upper part in a way I have seldom seen. One sees it commonly enough in the inner parts of the spaces, but I have not often seen it so widely spread. There was much impairment of resonance over all this area, as if the lung were absent. These signs made me think that the lung had retracted owing to old pleurisy, and that the two layers of pleura had adhered. That condition, together with adherent pericardium, accounted, I thought, both for the retraction of the spaces during systole and for the increase of precordial dulness.

What was the origin of all this? There was some suspicion of cancer, but I thought it was probable, owing to the condition of the lungs, that the disease was tuberculous, and that along with the pericardial adhesion, pleurisy, and chronic peritonitis which we thought existed there must, from the dilated venules in the head and neck, be fibrosis in the mediastinum as in the previous case. In making that diagnosis I had to face the fact that in the surgical wards he had been injected with tuberculin, both the old and the new forms, but with no tuberculin reaction, and that both there and later when he came to my wards the sputum was repeatedly examined for tubercle bacilli, but none were found. But he had a very hectic temperature in the surgical ward, and I thought the tuberculin reaction might have been masked by the hectic temperature which naturally belonged to him. And as far as the presence of tubercle bacilli goes, if you do not find them the evidence is at best only negative and you must never let it outweigh positive clinical evidence. So I continued in my opinion that he was tubercular. And that proved also to be the case. We found on post mortem a very interesting condition of things, namely that he had, as I thought, chronic peritonitis of a

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tuberculous nature, that he had adherent pleura all over his left side, and that, just as we thought, the two layers of pleura over the heart were firmly adherent, the lung being retracted and the heart being uncovered, and that he had a recent pleurisy on the other side, with effusion. He had also, as we expected, fibrosis in his mediastinum, which had pressed upon the great veins and produced cyanosis of head and neck. But there was an interesting point in which I was quite wrong, namely, there was no adhesion of pericardium whatever. It was quite a natural pericardium. And this shows that the systolic retraction of upper spaces, which is so suggestive of adherent pericardium that we always expect to find adherent pericardium when it is present, can exist without any such thing at all. In this case the negative pressure in the pericardium pulling upon the thickened tissues which adhered to it in front was sufficient to pull back the spaces at the time the apex came forward, without any adhesion at all between the parietal and visceral layers of the pericardium.

Both those cases were alike in this, that apart from the conditions of the heart and lungs respectively they had an indurated fibrosis of the tissues in the mediastinum, which in each case pressed upon the great veins leading back to the heart and produced the symptoms of cyanosis and dropsy of the abdomen. That has been called by a special name, "mediastinitis," a name which was given to it twenty years ago by Dr. Harris, of Manchester, and now chronic indurative mediastinitis finds a place in most text-books as a definite group of symptoms. But do please remember that it is not a new disease nor a separate disease because you give it a separate name. There is no objection to using a name if it gives you comfort. After all, Mesopotamia has its analogues in medicine as well as in religion. There is no reason why you should not use the name "mediastinitis" for the symptoms I have mentioned. There are a few cases in which the fibrosis which you find in the mediastinum begins there from suppuration or possibly tuberculosis of mediastinal glands, or perhaps from some other rare disease. But in the great majority of cases it is merely an extension from an adhesive pleurisy or adhesive pericarditis, and very often there are both.

So the lesson to be learnt is not that these things have a separate name and are a separate condition, but that if an obvious cardiac or pulmonary disease does not fully account for the symptoms which your patient exhibits do not be content to say it is merely an accident, but consider whether it cannot be explained by the extension of adhesive inflammation to the surrounding structures.

July 2nd, 1906.

LECTURES

ON

THE EXAMINATION AND TREATMENT OF EAR DISEASES.

Delivered at St. Bartholomew's Hospital.

By A. E. CUMBERBATCH, M.B., F.R.C.S.,
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LECTURE I.

GENTLEMEN,—I want to show you to-day how to examine an ear, because, naturally, until you know how to do so you cannot tell whether a patient is deaf or what his deafness is due to. It is, of course, essential that you should have a good light, and daylight is the best, chiefly because it is white light. If you are looking at an ear by artificial light its yellowness is very apt to alter the colour of the drum. If the membrane is very thin you will see a pinkish hue, which is nothing more than the reflection of the mucous membrane of the tympanic cavity showing through, leading you to think that the membrana is congested when it is not. You will understand what I mean if you look between your fingers held in front of a candle. But of course we cannot always get daylight in this country, and therefore, in the winter especially, we have to use the best artificial light which we can get. The patient should be seated between you and the light. You can use either a frontal or a hand mirror. Having looked, in the first instance, at the pinna, you satisfy yourself whether or not it is normal. Occasionally you will find that it is abnormal in one respect or another. In some cases the pinna falls forward, and an anxious mother wants to know what can be done to overcome the unsightliness. I may say in passing that the deformity may sometimes be remedied by strapping back the pinna for a considerable time or by removing the skin from a small area at the back of the pinna, and from a corresponding spot over the mastoid, and stitching the two surfaces together. Your procedure you will, of course, settle according to the degree of deformity. Sometimes the pinna is represented by only a nodule of cartilage. Often associated with this arrested development of the pinna you will find the meatus is occluded, and then the question will arise, Is the meatus normal,

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but covered by a diaphragm of skin, or is it undeveloped? Often that question can be answered by using the probe, but most likely only after an incision through the skin. Another abnormal condition of the pinna met with is hæmatoma. It may occur spontaneously, but is usually the result of an injury, generally received at football. It is caused by an effusion of blood between the perichondrium and the cartilage, on the anterior surface of the auricle, which becomes swollen to an unsightly extent, and is of a livid hue.

Another common affection of the pinna is eczema. Lastly, you may find chalk-stones, or an epitheliomatous ulceration of the outer ear. Those are details, more involving general surgery than this particular branch. The next step is to examine the meatus, and to do this properly grasp the auricle between the middle and index fingers, the speculum between the index finger and thumb, and pulling the auricle upwards and backwards, insert the speculum with a gentle rotatory movement. At a certain point you feel a resistance between the bony and the cartilaginous meatus, and beyond that you must not attempt to force the speculum, or it will cause pain. Next throw your light down the speculum and see if there is anything obstructing your view of the meatus and the membrana. The most common cause of obstruction there is, of course, cerumen, which appears as a blackish or brownish mass; or there may be an accumulation of epithelial *débris* or an excessive growth of hair just at the orifice of the meatus. Sometimes there is a polypus, or the meatus may be more or less occluded by a bony enlargement, or hyperostosis. Very often you cannot get your speculum in properly because there is a relaxation of the ligament which attaches the pinna behind to the bone, and the posterior wall falls forward. So instead of the meatus being circular, or nearly so, it is a contracted oval in shape. The difficulty is easily overcome if you pull the meatus well upwards and backwards, and inserting the speculum, coax it forwards so as to ride over the obstruction. It is merely slit-like at the orifice, and as soon as you have got beyond that you will be able to obtain a good view. In cases of inflammation of the meatus you will find that the passage is red, perhaps somewhat narrowed, and there is a certain amount of moisture—a thin, purulent discharge. On the other hand, there may be inflammation and suppuration of the middle ear, and you will then find muco-pus in the meatus. At other times you will find that your view of the membrana is obscured by polypoid granulations, or there may be a small granulation on some part of the membrana, or you may find that there is a perforation of the membrana. On the other hand, there may be calcareous deposits in the membrana, or perhaps part of the membrana destroyed and the remainder attached by adhesions to the inner wall of tympanum, all of which appearances you have to take notice of during your examination. But we will imagine you have examined the meatus thoroughly and found that there is nothing abnormal there. Now as to the appearances presented by a healthy membrana. You will notice that it stretches across the bottom of the meatus as a thin film, highly polished, of a greyish-yellow or greyish-blue colour, according to its thickness. If the membrana is very thin, the prevailing tint is blue-grey; when it is thicker it is more of a yellowish-grey, just as in the case of teeth. When the enamel of teeth is very thick, the teeth have a yellowish colour, whereas if it is very thin they are bluer. You will notice that the membrane is not placed straight before you, but obliquely; in fact, it has a double obliquity, forward and inwards. If you measure the meatal walls you will find the posterior superior wall is shortest, the anterior and inferior wall being the longest. In looking at this membrane the first thing which will catch your eye, or the first thing which you should look for, is that little point which you will see here. It is always prominent unless there be extreme thickening of the membrana. The spot is called the processus brevis, and it is a landmark to which you can refer all the other points of the membrana. It looks almost like a small sebaceous pimple, such as you can see on the skin of the face. From the processus brevis you run your eye downwards, and you can then see a white line, which is the handle of the malleus. This runs downwards and backwards to about the centre of the membrane, and then ends in a spatular enlargement, not very well shown here, at the most incurved part of the membrana, called the umbo. From the umbo a fan-shaped cone of light is thrown forwards and downwards, with its apex at the umbo and its base at the periphery. The cone of light is not of much importance except as showing the angle at which

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the membrane is placed. But when the membrane is much drawn in, as it is when the Eustachian tube is obstructed, the tendency of that cone is to be more horizontal, so that when looking at the membrana in chronic catarrh of the middle ear, in which the Eustachian tube is completely blocked, instead of the cone of light being downwards and forwards, it is more like this, thrown horizontally forward. Sometimes, owing to adhesions or thickening of the membrana, its surface is not smooth, but irregular, and therefore, instead of the cone of light being fan-shaped, you will see two or three spots of light, the membrana between them being in shadow. These simply indicate an abnormal position of the membrane. Now pass your eye backwards again to the processus brevis, and you will see two lines running backwards and forwards to the periphery, called the anterior and posterior ligaments of von Tröltsch. They are not of any particular importance, but if they are defined they mean that the membrane is altogether more indrawn than it should be. All the membrane above those two ligaments is called Shrapnell's membrane, or the membrana flaccida, and closes in the top of the tympanic cavity, which goes by the name of the attic. The attic is important because it is separated from the cranial cavity only by a thin layer of bone, and suppuration in this situation is especially liable to be dangerous. The membrana flaccida differs in structure from the rest of the membrana tympani in that it consists only of thin, irregular fibres of connective tissue, while the rest of the membrane consists of two layers of fibres, radiating and circular, covered within by the mucous lining of the tympanic cavity and outside by the skin lining the meatus. There is no design or arrangement of fibres in the membrana flaccida. It is much thinner and looser—hence its name—and it is more easily perforated. You will see a shadowy line here, which is the long process of the incus. In an ordinary ear you do not see that, but if you have strong daylight and the membrane is very thin, or the whole of it is indrawn, so that the membrane presses against it, you can readily see this long process of the incus. Therefore, when you see the long process it probably means that the membrana is too indrawn, because in its normal condition it does not touch this process. It is attached by the os orbiculare to the stapes. Having made your notes about the condition of the membrane, the next thing is to see whether air enters the cavity freely or not through the Eustachian tube, because middle-ear deafness generally begins with a post-nasal catarrh, which spreads up the Eustachian tube and causes obstruction to the free entrance of air. The tympanic cavity is practically a bony cavity, except externally, where it is closed by the membrana tympani. If that membrane is to respond freely to all the vibrations which impinge on it, there must be a perfect balance of pressure inside and outside this cavity. If the Eustachian tube is obstructed, there is a negative pressure inside the tympanic cavity and the membrana is driven in, some deafness ensuing. If you hold your nose tightly and swallow, you will notice that a sort of cloudiness or indistinctness of hearing occurs; that is because momentarily you have produced diminished atmospheric pressure within the tympanic cavity. It does not render you deaf, but you are conscious you do not hear sounds as sharply as before the experiment. There are three methods of forcing air into the tympanic cavity. The first is known as Valsalva's method. The patient holds his nose and then breathes forcibly, when, if the tube is patent, the air enters the tympanic cavity. To ascertain this fact, put one end of this tube, which is called a diagnostic tube, into your meatus and the other into the patient's, and if the Eustachian tube is patent you hear a sound which is best described as being like closing the lips together, and then opening them suddenly, or like the sound made by the letter "p" in the word "part." If there is complete obstruction, you hear nothing. If the Eustachian tube is blocked just at the junction between the bone and the cartilages, as it often is, and you listen carefully, you will hear the air strike the obstruction, only the sound is fainter and more distant. The next way of inflating the tympanic cavity, both for treatment and for diagnostic purposes, is by means of Politzer's bag. Put the tube into the patient's nostril, carefully close up his nose so that no air can get back, and then ask him to swallow. As he swallows squeeze the bag quickly. Many people find it difficult to swallow if they have nothing in the mouth, and therefore it is a good plan to give the patient a mouthful of water. If you use the diagnostic tube, you can hear whether air enters the tympanic cavity. Some people are very stupid about swallowing and breathing. Many

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go through life who cannot swallow at a given moment, just as there are many people going through life who never blow their nose properly. Many nervous people find it impossible to swallow just when you want them to; they either swallow before you have a chance of squeezing the bag, or else they swallow too late, and as you squeeze the bag you are somewhat alarmed by seeing them struggling for breath, because some of the water has gone down the trachea and some up the nose. A simpler way with such patients is to make them fill the buccal cavity with air, and then you squeeze the bag; by adding the air in the bag to that already in the buccal cavity the pressure is increased, and that is often sufficient to overcome the Eustachian obstruction. It is common, however, for a patient, when told to fill his mouth with air, to blow for all he is worth, by which means he is just defeating your object, as he is blowing air out of his nose, and by so doing is obstructing the air you are trying to force in. But you can feel in a moment if he is doing so by the distension of the bag. A good plan is to tell the patient to imagine he is about to blow out a candle, and prepare to do so, but instead of actually blowing, hold the air in his mouth by shutting the lips tightly. If you fail by this plan—and many are too stupid to do even this correctly—another way is to make the patient say "hick, haec, huck"; sounding the guttural tends to lift the palate and open the Eustachian tube, and as he says "huck," squeeze. That is the most perfect way if there is only a slight obstruction. But where there is much constriction of the Eustachian tube you cannot get air into the tympanic cavity by this method so satisfactorily as by swallowing water or by distending the cheeks.

If Politzer's bag fails, then you must pass the Eustachian catheter. There are three chief methods for passing the catheter.

First method.—Tilt up the end of the nose with the thumb, and holding the catheter very lightly between the right thumb and forefinger, pass it quickly along the floor of the inferior meatus, slightly depressing the shaft of the catheter till the point of the instrument is felt against the posterior wall of the pharynx. Rotate the instrument outwards, slightly withdrawing it till the point is felt to ride over the posterior lip of the Eustachian orifice; then rotate further till the ring on the under part of the base of the catheter is in a line with the outer canthus of the eye. Press the instrument outwards and onwards, and the point will enter the Eustachian tube. It can then be held in position by the left forefinger and thumb. I show you the method here on this cadaver.

The second method is to pass the catheter as before, then draw it forward till it is felt to ride over the posterior edge of the palate, turn outwards till the ring is in a line with the outer canthus of the eye, then push the point into the orifice of the tube as before.

The third method is to hook the concavity of the catheter round the posterior edge of the vomer, then rotate it downwards, outwards, and slightly upwards, and push it onwards into the tube. This method is quite impracticable if the palate is very irritable, or if there is much œdema or thickening of the posterior part of the pharynx. In many cases it is extremely difficult, and sometimes impossible, to pass the catheter by any method. Such is the case if there is much deviation of the septum or enlargement of the posterior end of the inferior turbinate bone. Sometimes by manipulating the instrument and coaxing it round or under the obstruction or by altering the curve, it may be successfully passed when, at a first attempt, it seems impossible. Painting the inferior meatus with solution of cocaine will lessen the difficulty due to an irritable condition of the pharynx. As a last resource the curve of the catheter may be increased and an attempt made to reach the tube from the other inferior meatus. In all cases extreme gentleness must be used, as any force may tear the mucous membrane near the Eustachian orifice, and inflation may then be followed by emphysema around the glottis and suffocation.

Having settled the question as to the patency of the Eustachian tube, the question is what you hear on listening with the diagnostic tube. If the tympanic cavity is dry you hear the sound I have already mentioned. If it is obstructed and you listen carefully you may hear the air impinging at some point short of the tympanum. If there is fluid in the tympanic cavity you will hear a gurgling sound, very much like the moist sounds in a cavity in the lung. It is, of course, much fainter, but there is the same bubbling sound which air makes when it churns up mucus in a cavity. But if there is perforation, the air not being checked by the

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membrana, whistles through the opening and through the diagnostic tube, and then it sounds to you exactly as if the air had been blown directly into your own meatus. The next point is to test the patient's hearing. The simplest thing to work with for that purpose is a watch with a rather loud tick. Commence by bringing it from a distance to his ear—(do not commence near to his ear and gradually withdraw it)—and ask him to tell you when he hears it. You should previously ascertain at what distance you can hear the watch ticking yourself. Supposing it is thirty-six inches, you use that as the denominator, and the distance at which the patient can hear it as the numerator. Thus, if the patient can hear your watch no further than four inches off, your note will read 4/36. While testing, make the patient close the sound ear. There is an instrument called the acoumeter, invented by Politzer, which you can use instead of a watch. I do not advise its use, however, as it is rather a fragile instrument.

The next procedure is to test the hearing power for the voice. Pitch your voice to a particular intensity and then, standing back, ask the patient certain questions which he is not likely to guess at or understand if he does not hear them properly. You may ask him to repeat the questions which you ask him. Then come gradually nearer and notice the distance at which he clearly hears your voice, which is still pitched in the same key. Then try him with whispers. The next point you must settle is as to whether his deafness is entirely due to middle-ear catarrh or to some affection of the labyrinth, auditory nerve, or auditory centres. For that purpose one employs the tuning-fork. When a vibrating tuning-fork is placed on top of the head in the middle line, it is heard by a person of normal hearing, and the sound is referred to the point of contact. If now one meatus is closed the sound is heard louder in the closed ear. The explanation given, which I believe to be correct, is that the vibrations which have reached the labyrinths through the cranial bones, escaping through the line of least resistance, the meatus, are thrown back, in the closed meatus, and at their points of intersection are intensified. You know that if a stone is thrown into still water a series of concentric circles form, and if a second stone is dropped near the first, a second number of circles form, and where these waves intersect they are higher than elsewhere. The same law applies to sound.

The act of closing the meatus by making the membrana more rigid, also assists in preventing the free escape of the waves of sound. As the vibrations are intensified in the closed ear, the nerve-centres refer the sound to that ear instead of referring it to the point of contact. Whether this explanation be correct or not, the fact remains, as you can easily test for yourself. This tuning-fork test is used to diagnose between middle-ear and internal-ear deafness, or nerve-deafness as it is generally called. Suppose, for instance, a patient whose right ear is deaf says he hears the tuning-fork when placed in the middle line of the vertex, louder in the right ear, you conclude that the deafness is due to middle-ear trouble. Owing to thickening and rigidity of the membrana tympani and probably of the membrana closing the fenestra rotunda, the vibrations do not escape so freely through the right meatus as through the left, and the sound is therefore louder in the right ear. If, however, he hears the fork louder in the left ear (the sound one), you infer that the deafness is due to some affection of the labyrinth (for present purposes we will exclude the auditory nerve and cerebral centres), because the vibrations carried to the right labyrinth fail to make as strong an impression on the nerve-terminals of the right labyrinth as on those of the left, and the sound is therefore referred to the left ear. This is Weber's test.

In normal hearing, when a vibrating tuning-fork, placed on the mastoid process, has ceased to be heard, if the prongs are placed opposite and quite close to the meatus, the sound can still be heard for some time, because aerial conduction is better than bone-conduction. If, therefore, a patient who is deaf in the right ear hears the tuning-fork opposite the meatus after he has ceased to hear it on the mastoid, we conclude that the affection is in the labyrinth (nerve-deafness), whereas if he cannot hear it we infer that the affection is in the middle ear. The explanation is this: as aerial conduction is, as I have just said, better than bone-conduction, if there is no thickening of the membrana tympani, or the lesser membrane closing the fenestra rotunda, the vibrations of the tuning-fork pass through the tympanum to the labyrinth and are readily perceived, whereas

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if there is thickening of the tympanic structures the faint vibrations are so impeded that if they do reach the labyrinth they are too feeble to make any impression on the terminal fibrils of the auditory nerve. This is Rinné's test. When the tuning-fork can be heard opposite the meatus after it has ceased to be heard on the mastoid, we say, for brevity's sake, "Rinné positive"; when it cannot be heard opposite the meatus after it has ceased to be heard on the mastoid, we say, "Rinné negative." It often happens that both tympanum and labyrinth are affected, and the amount of disease in both may so balance each other that the patient is unable to decide whether he hears the tuning-fork louder in the sound or the affected ear. To settle the question transfer the fork quickly from the mastoid of the deaf ear to your own mastoid after the patient has ceased to hear it. If his labyrinth is sound, it is evident, from what has been already said, that you will be unable to hear it also. If, therefore, you can hear it an appreciable time after he has ceased to do so, it is evident there must be some affection of his labyrinth. By your earlier examination you may have decided that the tympanum is affected; you will, therefore, conclude that the deafness is tympano-labyrinthine. Of course through a preconceived idea, or through stupidity, a patient may give incorrect answers, but a little practice will in most instances enable you to detect when this is so.

Now, you must not imagine that everything I have told you about the tuning-fork tests is mathematically correct. Every now and again you will not be able to square your facts with your theories. Rinné's test, for instance, is sometimes puzzling, but in the great majority of cases there is not much trouble in arriving at a correct diagnosis.

Having finished testing the hearing, the next thing is to examine the naso-pharynx. I always go through that routine, because you must remember that probably 90 per cent. of the cases of ear trouble apparently proceed from the naso-pharynx, up the Eustachian tube. Find out whether the patient has had syphilis or tubercle, or a family history of either, or whether deafness is a family inheritance. Also inquire whether the patient has been bathing and got some sea water into his ear; the answers to these questions may give you valuable indications as to the kind of treatment you should adopt.

I have some patients waiting in the out-patient department who will illustrate some of the points we have been discussing.

July 2nd, 1906.

WITH
MR. H. L. BARNARD
AT THE
MEDICAL GRADUATES' COLLEGE AND POLYCLINIC.

THE ANATOMY AND PHYSIOLOGY OF THE STOMACH.

GENTLEMEN,—It is always a sound principle to commence the consideration of the diseases of any organ with a review of the anatomy and physiology of that organ, especially of the more recent advances on these subjects. We shall therefore review to-day those parts of the development, anatomy and physiology of the stomach which seem most clearly to elucidate the nature and treatment of its diseases.

(1) DEVELOPMENT.—The stomach in its early stages of development is a medium organ in which the anterior border to which the gastro-hepatic omentum is attached looks directly forward, whilst the posterior border and the gastro-splenic omentum are behind. It is the rapid growth of the great curvature and the budding out of the cardiac fundus that lead to the rotation of the organ, so that its posterior border passes to the left and its anterior border, although to a much less degree, is displaced to the right. It will at once be seen that the left surface of the fœtal stomach thus becomes anterior and the right posterior. The application of this fact to surgery is in connection with referred pains. There is some evidence, although perhaps not conclusive evidence, to show that the referred pain from an ulcer on the anterior wall of the stomach is more marked on the left side and of one on the posterior wall on the right side of the back and epigastrium. As Mr. Arthur Keith has pointed out, the cardiac fundus sprouts out from the stomach in a very similar way to that in which the cæcum buds out from the colon. This separate development appears to indicate a separate function for this part of the stomach, and the cardiac fundus certainly has a separate blood and lymphatic supply. The oxyntic or border cells are more numerous in the glands of the cardiac fundus, and the study of the movements of the stomach shows that the fundus is chiefly used as a reservoir from

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which the actively churning pyloric portion is fed. We shall see when we come to discuss the diseases of the stomach that ulcer is very rare on the cardiac fundus, perhaps because the food is not actively churned up in this part. The separate lymphatic supply of the cardia has an important bearing on cancer; for in removing a cancer of the pyloric part it is practically safe to leave the fundus, in which there will probably be no secondary deposits. Conversely cancer of the fundus, when it does occur, which is seldom, does not tend to spread to the rest of the organ, and is peculiarly liable to lead to cancerous enlargement of the glands of the left posterior triangle of the neck.

(2) ANATOMY.—The position and axis of the stomach have been the subject of much misapprehension, apparently because the position of the stomach was studied after death, when the diaphragm was in a condition of complete expiration, and the abdominal wall was either completely relaxed or entirely removed, and the stom ch itself was in a condition of paralytic dilatation. Observations on the living during operation, or by means of bismuth emulsion and the Rontgen rays, indicate that during life the stomach is nearly vertically placed. Its cardiac orifice is just to the left of the middle line, under the seventh left costal cartilage. The pylorus is just to the right, about a hand's breadth lower down. The two orifices are only separated from one another laterally by the width of the sternum. Many people consider that the stomach is placed transversely across the abdomen; but this is, of course, an error, as the cardiac orifice is deeply placed just to the left of the spine, whilst the pylorus is nearer the anterior abdominal wall, so that the axis is obliquely placed and directed from behind and the left down forwards and to the right. The small curve and the orifices are, moreover, nearly fixed, whilst the fundus and great curve are capable of free movement, and it is by enlargement along these borders that increase of capacity within a wide range is provided for.

The relations of the stomach.—The organs which are in contact with the anterior and posterior surfaces of the stomach are those which will become adherent or ulcerated. In front the stomach is in contact with the liver, diaphragm, and abdominal wall. When an ulcer perforates the area in contact with the liver, it is frequently sealed off by adhesions between the two. This seldom occurs in the two areas in contact with the diaphragm and abdominal wall, presumably because these parts are in constant and active movements. The posterior relations of the stomach are chiefly of surgical interest, because large chronic ulcers frequently remove large areas of the stomach wall and leave these organs exposed in the floor of the ulcer. I show you here such a specimen in which the pancreas is exposed over an area the size of a five-shilling piece. The anterior wall of the stomach looks towards the greater sac of the peritoneum, and perforation there will involve that cavity, whilst the posterior wall is in the lesser sac of the peritoneum, and a perforation will first involve the lesser sack and may not spread farther if the foramen of Winslow be sealed off. The second part of the duodenum is placed immediately over the hilum of the right kidney to which it is attached. When the kidney slips downwards and rotates forward on the renal artery, the second part of the duodenum may be twisted and kinked so as to obstruct its lumen. The stomach will then become dilated. This association of dilated stomach with right movable kidney has long been recognised. A case of this sort was transferred to me at the London Hospital by Dr. Robert Hutchison. She was a woman of about thirty-five years of age, with typical dropping of the stomach. The lesser curve was clearly felt and seen below the liver margin and the greater curve descended far below the umbilicus when she stood up. She had all the symptoms of dilated stomach—vomiting at long intervals of great quantities of fermenting food, and progressive wasting. The interesting point, however, was that when she was put to bed, and the foot of the bed raised the stomach emptied itself, and she rapidly put on flesh. After this had happened once or twice, with relapses as soon as she stood up, Dr. Hutchison transferred her to me for gastro-plication.

On examining her, however, I found that the right kidney was excessively movable, and decided to fix that first as this operation involved very little risk. The result was most satisfactory, and she was then able to stand up without kinking her duodenum and dilating her stomach.

The arteries in relation with the stomach are of surgical importance not only because ulceration leads to hæmorrhage from them, but also because their position must be known in order that they may be dealt with in operations on the viscus.

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The coronary splenic and pancreatico-duodenal are those from which arterial hæmatemesis most frequently occurs. The coronary artery is not only in relation with that part of the stomach which is most frequently ulcerated, but it is in contrast with the gastro-epiploic arteries in very close contact with the stomach wall, no doubt because the lesser curve is a fixed point and the greater curve advances and recedes between the layers of the greater omentum. These facts together account for the great frequency of hæmorrhage from this artery. The splenic artery is exposed by ulcers of the posterior wall which lay bare the pancreas, whilst the pancreatico-duodenal artery is in contact with the posterior surface of the pyloric antrum—a very common site of ulcer.

The veins of the stomach present several points of surgical interest. Very rarely hæmatemesis, in cases of gastric ulcer, occurs from them, but they so readily thrombose in this condition that this seldom occurs.

In cirrhosis of the liver, however, when the obstructed portal vein is engorged with blood, the most profuse and rapidly fatal hæmorrhages may occur from very numerous points on the gastric mucous membrane. In some cases, however, no such point can be found from which the blood has issued, and then it may be explained in some cases by supposing that the hæmorrhage took place from the large collateral veins which pass through the cardiac sphincter into the œsophageal veins. In advanced cases of cirrhosis these veins are said to hang like bunches of distended piles into the stomach. When the patient has bled to death the veins collapse and withdraw into the œsophagus, and the minute puncture from which the blood issued is overlooked.

The third point of interest about gastric veins is their possible association with post-operative hæmatemesis. In many of the recorded cases in which severe and even fatal hæmorrhage has followed an abdominal operation the omentum has been ligatured or severely handled, and it is claimed by some that thrombosis spreads up its veins to the gastro-epiploic vessels and leads to thrombosis in the gastric veins, and thence to hæmorrhage either from consequent congestion or digestion of the areas involved, but this theory, though ingenious, lacks actual proof. In many instances the omentum has not been injured at all.

The lymphatic tissue in the mucous membrane of the stomach is aggregated into follicles, and is, according to most accounts, more abundant in the pyloric portion than in the body. This fact is perhaps connected with the greater frequency of ulceration there. According to Mr. Arthur Keith lymphoid tissue is found in larger quantities in the cardiac fundus.

Dr. Charles Miller was kind enough to show me sections from all parts of the stomach the diseases of which he is investigating, and from these sections it is clear that the chief aggregation of lymphoid tissue in the submucous tissue of the stomach is in the pyloric region. A less extensive massing of lymphoid follicles is found in the cardiac fundus. Lymphoid follicles are only found at long and irregular intervals throughout the body of the stomach. In the pyloric regions these little rounded masses of adenoid tissue lie between the bases of the pyloric glands at regular and frequent intervals and become more numerous as the pyloric sphincter is approached. In only one or two instances did Dr. Miller show me lymphoid follicles on the surface like a Peyer's patch, and then there was doubt that the mucous membrane had been removed by ulceration or post-mortem digestion. These lymphoid follicles are probably foci from which sepsis and acute ulcers of the stomach commence.

The lymphatic vessels are divided into three main groups, and roughly may be said to follow the veins in position and direction. Their surgical importance is that they determine the direction and limits of cancerous extension.

The most important group runs from the pylorus along the lesser curve accompanying the coronary vein. These lymphatic vessels and glands are intimately connected with the stomach wall, and it is necessary to remove this as high as the point at which the coronary vessels reach it in order to effectually clear out cancer of the pylorus. These coronary lymphatics terminate in the cœliac glands in front of the aorta.

The second set runs from left to right along the greater curve accompanying the right gastro-epiploic vein. This group is not in contact with the stomach wall, and to include it with the part removed in a partial gastrectomy it is necessary to remove rather more than an inch of the omentum attached to the stomach. These lymphatic vessels

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terminate in glands below the pylorus, and behind it around the head of the pancreas, and these must be carefully removed in cases of cancer.

The third set of gastric-lymphatic vessels converge from the cardiac fundus and the left extremity of the greater curve, and pass through the gastro-splenic omentum to join the splenic lymphatic glands.

Another point of great surgical importance is that the lymphatics do not pass from the pylorus towards the duodenum, so that the section may be made only an inch beyond the obvious limit of a pyloric cancer, whilst from what has been already said it will be clear that the section through the body must be made in a line from the œsophagus to a point to the left of the middle of the greater curve.

The nerves of the stomach are derived from the two vagi, and from the sixth, seventh, eighth, and ninth dorsal segments of the spinal cord through the sympathetic system.

As has already been pointed out, under the head of Development, the referred pains from the anterior surface tend to be on the left side, and those from the posterior surface on the right. Moreover the cardiac orifice is innervated from the sixth and seventh dorsal segments, and the referred pain and hyperæsthesia from an ulcer in this region will be in the skin-distribution of these segments. The pylorus is innervated by the ninth dorsal segment, and an ulcer in this region, as is commonly the case, will refer its pain in the corresponding skin-segment.

The gastric glands are of two types, the pyloric and the cardiac. In the pyloric glands the duct leading to the secreting acini is much deeper. The secreting acini are several for each duct, and the cells lining them are all of one type and are believed to secrete pepsin. In the cardiac glands the duct is much shorter. The secreting portion of the gland is usually formed of a single tubule and is lined with central cells, similar to those in the pyloric glands. There are, however, in addition border or oxyntic cells which secrete the hydrochloric acid. These cells are most numerous in the glands of the cardiac fundus. The microscopic character of the gastric glands seems to have very little influence over gastric diseases. It is, however, stated that in advanced gastric cancer there is a general degeneration of all the gastric glands, and it is certain that hydrochloric acid and pepsin disappear from the gastric contents.

(3) PHYSIOLOGY. — The physiology of the stomach is best considered under the heads of—

(A) Gastric movements.

(B) The secretion of gastric juice.

(C) Absorption from the stomach.

(A) *Gastric movements.*—The movements of the stomach have been very fully investigated by W. B. Cannon ('American Journal of Physiology,' vol. i, 1898, p. 359), where a very full review of the literature of this matter will be found. Cannon's method was to feed cats on 15 grammes of bread and milk to which 5 grammes of bismuth subnitrate were added. He then watched the movements of the stomach by means of X rays and a fluorescent screen. Female cats were much more tolerant of the restraint of the cat-holder than tom-cats.

(1) *Movements of digestion* may be divided into (*a*) churning movements and (*b*) the evacuation of the stomach contents.

(*a*) *Churning movements.*—Cannon found that five minutes after a meal had entered the stomach peristaltic waves appeared as constrictions in the gastric shadow near the pylorus and progressed slowly to the right. Gradually these constrictions commenced farther and farther to the left, until they all started from the transverse band—that is to say, a constriction about the middle of the stomach. This band, which is an anatomical feature of the stomach, becomes more and more tonic as digestion proceeds, but does not actually divide the lumen of the stomach in two until the end of digestion. To the left of the transverse band the cardiac part of the stomach acts only as a reservoir and undergoes no change except a general tonic contraction. The pyloric part of the stomach to the right of the transverse band is more or less clearly divided into the pyloric antrum and the pre-antral part. When digestion is thoroughly established a constriction starts every ten seconds from the transverse band and in thirty-five seconds reaches the pylorus. There are always, therefore, three or four constrictions in transit in the pyloric part of the stomach. Cannon estimated that 2600 peristaltic waves were employed by a cat in the digestion of an ordinary meal of bread and milk. These churning movements of the stomach are at once inhibited by pain or emotional excitement of any sort. As

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digestion proceeds the pyloric part of the stomach becomes more and more elongated. The control of the pyloric sphincter is a most interesting matter; it is controlled partly from the stomach above and partly from the duodenum below. Cannon gave his cats little buttons of hard-baked dough nearly solid with bismuth. He was able to watch these approach the pyloric sphincter and strike against it. The sphincter was then closed during several contractions and no further chyme escaped into the duodenum. It has long been known that solid masses are not allowed to pass the pylorus until the end of digestion, when the stomach completely empties itself. This reflex is, no doubt, much more sensitive when an open ulcer exposes bare nerve-terminals, and in these cases spasmodic contraction of the pylorus is almost constant.

It was Pawlow who found that an even greater control was exerted over the pylorus by the reaction of the duodenum, and this has been named the acid reflex of Pawlow. Whilst digestion was actively proceeding he was able through a duodenal fistula to introduce acids and alkalies into the duodenum. He found that when acids were introduced the pylorus was closed, and no more acid chyme was admitted to the duodenum, whilst when the duodenal contents were rendered alkaline nearly every gastric contraction forced chyme into the duodenum. This is, no doubt, due to the fact that in an acid medium pepsin will digest trypsin, and that were the duodenum flooded with acid chyme the whole of the pancreatic digestion would be disorganised. By the acid reflex the chyme is only admitted at just the rate at which it can be neutralised. There is also some evidence that mechanical distension of the duodenum closes the pylorus reflexly.

(*b*) *The evacuation of the stomach contents.*—Pawlow found that when the stomach was empty at intervals peristaltic waves passed along it towards the pylorus. When food was admitted to the stomach these movements of evacuation were inhibited, the pylorus contracted, and churning movements appeared. He further found that the administration of acids increased the pyloric contraction and retained the gastric contents, whilst alkalies especially sodium bicarbonate, relaxed the pylorus, and led to rapid evacuation of the food in the stomach. It is clear from this that if we wish to increase gastric work we should give acids, whilst if our object be to save as much as possible an inflamed organ we should soon after a meal administer an alkali, and thus shift the burden of digestion on to the pancreas.

(2) *Movements involved in vomiting.*—Cannon found that during vomiting the pylorus, transverse band, and the pyloric part of the stomach were tonically contracted. No doubt this contraction is relaxed between the spasm of continued emesis, for otherwise it is hard to explain why gastric contents are so constantly followed in all persistent vomiting by bile-stained fluid. When the pylorus has been firmly closed the cardiac sphincter is relaxed, and the contraction of the longitudinal fibres of the stomach draws the cardiac orifice open and disposes the cardiac reservoir in a convenient position below it for the expression of its contents. The actual ejectment of these contents appears to be effected by the simultaneous descent of the diaphragm above and the contraction of the abdominal wall below. These two muscular bodies squeeze the stomach between them, and force the contents up the œsophagus.

(B) *The secretion of the gastric juice.*—Our present complete knowledge of gastric secretion we owe almost entirely to Pawlow. He has utilised in physiology the great advances in antiseptic surgery and the technique of œsophageal and gastric surgery to establish in dogs permanant œsophageal, gastric, and duodenal fistulæ, and to isolate from the main cavity a miniature stomach which retained its blood and nerve supply intact, and was connected to the surface by a separate fistula. Direct observation showed that the secretion of this miniature stomach accurately followed that of the larger cavity, and differed only in being about one tenth in quantity. The great advantage was, of course, that the juice was not mixed with the food introduced into the larger cavity.

The discoveries of Pawlow and his pupils may be summarised thus: The secretion of gastric juice commences five minutes after the animal sees and desires food; and although the mastication and tasting of the food assists this flow, it is not necessary that the material should enter the mouth at all. This "psychic juice" as Pawlow named it, acts upon the food when it reaches the stomach and elaborates from each article of diet a specific excitant, which acts upon

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nerve-terminals in the mucous membrane and leads to a second secretion of gastric juice, which is exactly adapted in quantity and strength of ferment to digest that particular kind of food. This second secretion is therefore the result of a chemical stimulation of secretion.

(1) *The psychic juice.*—In order to demonstrate this secretion, Pawlow employed œsophagotomised and gastrotomised dogs. The food devoured and swallowed fell out of the fistula in the neck. The resulting secretion of gastric juice was collected from a tube placed in the gastric fistula, and was measured and investigated as to its digestive power. It was found that the flow commenced about five minutes after food was offered to the dog, and varied in quantity, rapidity of flow, and digestive strength with the desire the dog had for the food in question. Most curious and interesting differences were thus discovered. Not only was much juice secreted when the dog was hungry and little when he was satisfied, but the flow varied acording to the dog's palate. Thus meat excited a profuse flow, but bread and milk a much less secretion, and even individual peculiarities, such as a special liking for certain kinds of food, invariably produced a corresponding change in the gastric juice dropping from the gastric fistula. The co-ordination even went so far that when a dog had several times been teased in vain by the preparation of a tasty meal under his very nose, which was after all not given him to eat, he would at last learn the trick and turn away sulkily, and no juice or very little would then be secreted. This commencement of all gastric digestion by a purely psychic flow has, of course, a most important bearing on human medicine and health. For digestion to be complete and efficient the man must hunger for his food, it must be of such a kind as he likes, and he must eat it with attention and relish. The notion that the value of a diet depends on the number of calories of energy its constituents contain is obviously absurd. For food, however nutritious in theory, which is abhorred, introduced without appetite or even with repugnance into the stomach, will lie there an inert mass for an hour or more and ferment. The importance of attention, appetite, and leisure during meals is also obvious, and the bad habit of reading at meals must be strongly condemned.

(2) *The chemical stimulation of secretion.*—Pawlow investigated the manner in which the specific secretion for each article of food was brought about by employing dogs in whom a miniature stomach had been isolated from the main cavity. Whilst the dog slept various articles of diet were introduced through a fistula into the main stomach unnoticed by the animal, so that no psychic excretion was excited. The secretion resulting was collected from the miniature stomach uncontaminated by the food.

Pawlow found that articles of diet so tested might be divided into four classes—(*a*) those that produced a powerful secretion, (*b*) those that produced a moderate secretion, (*c*) those that produced no secretion at all, and (*d*) those that actually inhibited secretion.

The latter were discovered by mixing them with substances from classes (*a*) and (*b*), when they diminished or actually stopped the flow which would otherwise have resulted.

(*a*) *Substances which produce a powerful gastric secretion.*—These are meat extractives, such as soups, broths, beef-tea, but especially Liebig's extract. The secretion commences in about a quarter of an hour. This interesting demonstration of the powerful action of these substances clearly explains why soup is taken before the modern civilised dinner, which is for the most part eaten without much appetite, desire, or actual hunger, and is usually far beyond the actual needs of the organism.

It also elucidates the great benefit derived from mixing beef-tea or meat extracts with the food when a patient is very ill and has not the least appetite for food but the stomach itself is not diseased. When, however, the stomach is irritated, inflamed, or even ulcerated meat extractives are obviously contra-indicated, for our object should then be to give the organ all the rest we can.

(*b*) *Substances which produce a moderate gastric secretion.*—These are water, milk, and gelatine. It is not very clear why water should invariably produce a moderate and quite active secretion of gastric juice in the dog, unless it be that this fluid is so often dirty and contaminated that Nature has provided this gastric flow in order to kill and digest water-borne infections. Pawlow seems to have paid little attention to the antiseptic action of the stomach and duodenum; for it has long been known that although a phthisical patient must swallow myriads of tubercle bacilli, these cannot be

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found in his fæces until his ileum becomes ulcerated, and this does not occur until dyspepsia is well established. The automatic and moderate secretion of gastric juice for milk is, again, of great interest; for the new-born babe cannot take much interest in its diet, and universal empirical experience has shown that there is no diet so nutritious as milk for those who are too ill to desire food or take it with appetite or attention. The empirical use of jellies as an important part of all invalid diets is also hereby explained by the automatic and moderate secretion gelatine excites.

(*c*) *Substances which produce no gastric secretion.*—These are egg white, sodium chloride, starch, and sugar. These substances were, of course, given dissolved in water, and when it is said that they produced a negative result it is meant that the secretion was no greater than a corresponding quantity of plain water.

That raw white of egg should of all proteid substances require no gastric juice for its digestion is certainly a most interesting fact, especially when it is remembered that for many years it has been used as an even more suitable and less exacting diet than even milk in all cases of gastritis. The absence of all secretion in the case of starch and sugar is easily explained by the fact that gastric juice is much less adapted to the digestion of these than the pancreatic secretion.

(*d*) *Substances which inhibit gastric secretion.*—These are sodium bicarbonate and fats. We have already seen under "Movements of the Stomach" that sodium bicarbonate leads to a rapid evacuation of gastric contents through the pylorus into the duodenum, and to this we can now add that its administration checks further secretion, a most useful thing if it be excessive, and neutralises such juice as has already been secreted, thereby rendering the pepsin inert. We have, then, in sodium bicarbonate a most useful means of terminating gastric activity whenever we wish it.

When Pawlow poured oil into the stomachs of his dogs and soon after fed them on meat no gastric juice appeared for half an hour, and then the flow was feeble and of weak digestive power. Considering that the secretion which failed to appear was the psychic juice, this inhibitory action must have been a reflex nervous one, acting on the glands or their secreting centre from the mucous membrane of the stomach.

We see many examples of this powerful inhibition in dietary. The familiar combination of bread and butter throws very little pressure on gastric digestion, which is restrained by the butter, but when the combination passes into the duodenum the fat powerfully stimulates pancreatic secretion and the bread is digested with it. Bread alone requires a small quantity of powerful juice, and butyric acid powerfully stimulates gastric secretion, probably because butyric acid fermentation is inhibited by hydrochloric acid. Bread and rancid butter therefore are the very antipathy of bread and fresh butter in their effect on gastric digestion. The worst effects of the inhibitory action of fat on gastric secretion are seen when it is combined with meat and administered to an enfeebled stomach. Perhaps the most familiar example of this is when fat mutton is forced on anæmic girls by austere parents, and produces severe dyspepsia and vomiting.

It is in such cases of weak gastric digestion that meat nearly fat free, such as fowl and fish, should be employed. In cases of hyperacidity fatty emulsions may be used to restrain the secretion. Pawlow found that the moderate quantity and strength of the juice required to digest milk was due to the fat in it. When cream was added to the milk the resulting juice was even further diminished and weakened. When fat was removed from the milk the juice became more abundant and stronger.

To sum up the foregoing paragraphs. When an animal hungers and desires food, and more especially when his desires culminate in eating and swallowing food that he likes, the stomach by a reflex psychic act pours out gastric juice on that food within five minutes. This juice starts the process of digestion and elaborates from each article of food specific substances which act upon the nerve-terminals in the gastric mucous membrane and direct the course of gastric secretion so that the juice is exactly adapted to the food to be digested.

It thus appears that each kind of food has its own particular kind of juice, and that these vary in quantity and digestive strength. In the same dog the same food always produces the same juice in the same quantity. If the food is doubled the juice is doubled. From this it appears that in health secretion is most exactly adapted in quantity and quality to the work to be done, and there is

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a corollary to this that in health there is no excess of juice secreted to digest and irritate the gastric wall. Gastric juice is far too corrosive a fluid for Nature to slop it about an alimentary canal in careless excess.

We may here enumerate a few of the varieties of juices which were secreted from the miniature stomachs of Pawlow's dogs when they were fed on special articles of food. In these the psychic and chemical secretion are combined, as the animal was allowed to devour the food which passed immediately into the main cavity of its stomach.

(*a*) Meat produced a large quantity of gastric juice of weak digestive power, (*b*) bread produced a small quantity of powerful secretion, (*c*) milk a small quantity of small digestive power.

Milk is so important a food in disease as well as in health that its digestive characters may be reviewed and emphasised here.

In the first place its digestion requires no psychic juice to start it. Taken into the stomach, it acts immediately upon the the secretory nerves and produces its appropriate juice. It is therefore one of the only available foods for those who have no appetite or who, prostrated by disease, take no interest in food or any other thing. Nearly all other foods would under these circumstances fall into the stomach and lie there inert until they decomposed.

In the second place the amount and strength of juice required to digest milk is very small, so that the least possible call is made on a stomach the powers of which are already greatly lowered.

In the third place the proteid of milk is in a most available and easily digested form. If it be agreed that the increased secretion of urea after a meal is a gauge of the amount of proteid used up in the assimilation of the remainder, then in the case of milk 90 per cent. is assimilated, whilst only 50 per cent. of the proteid of bread is available under the same circumstances.

One or two further facts alone remain to be stated. Pawlow found that gastric juice was always secreted at a constant acidity of 0·5 per cent. of hydrochloric acid. It is well known from test meals that this acidity is reduced to a variable extent under different circumstances. No doubt this reduction is in part at least effected by the neutralising action of the food ; but probably there is some physiological means of reducing the acidity of the juice when once secreted to an optimum for the digestive processes proceeding. It is definitely known that an accumulation of hydrochloric acid stops the secretion of gastric juice.

When the stomach is empty its mucous membrane, like every other mucous membrane, secretes an alkaline viscid fluid. Pawlow found that during attacks of gastritis in his dogs even when empty the inflamed stomach secreted a thin acid fluid. When we consider the etiology of gastric ulcer we see that this observation is of great importance, for these alkaline intervals are probably of great importance for the repair of lesions of the gastric wall.

(c) *Gastric absorption.*—Apparently the stomach absorbs very little. When its digestive work is done or when it has none to do it passes its contents rapidly into the duodenum, from the villi of which rapid absorption occurs.

The only direct experiment on this point of which I am aware is that of Dr. Edkins. He ligatured the pylorus in a cat and then introduced into its stomach a many times lethal dose of strychnine, a drug to which cats succumb in the smallest doses. No ill result happened during several hours whilst the pylorus was ligatured, but as soon as the ligature was removed the cat passed into strychnine convulsions.

July 2nd, 1906.

Anæsthesia with the Roth-Dräger Apparatus.—Hagen reports very favourably on this method of inducing anæsthesia, basing his conclusions on seven hundred cases in which the apparatus was used. The procedure consists in the administration of a continuous stream of oxygen to which accurately measured quantities of ether or chloroform, or both together, may be added. The proportions of each constituent of the mixture are readily controllable. The advantages claimed for the apparatus are that it allows of extremely accurate dosage and of the widest possible range of individualisation. The anæsthetic vapours are administered in greatly diluted form and in reduced quantities. The accumulation of carbon dioxide in the body is prevented, and the stimulation produced by the oxygen is beneficial. Induction of anæsthesia is easy, and is usually unaccompanied by the stage of excitement ordinarily seen. The only drawback to the method is its expense, which is considerable.—*Medical Record*, vol. lxix, No. 23.

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TWO LECTURES

ON

TUBERCULOUS ARTHRITIS.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

LECTURE I.

GENTLEMEN,—I lecture to-day on tuberculous arthritis. I shall not complete to-day what I think ought to be said, so that on the next occasion I expect to lecture upon the same subject. I have chosen tuberculous arthritis for very simple reasons. I believe that under my care in this hospital I always have from sixty to seventy patients, and invariably from six to eight are suffering from tuberculous disease, a very high proportion, from 10 to 12 per cent. If you were to consider the records of the pathological department you would find—it has been found—that for every person who is systematically examined not less than 9 per cent. bear upon their bodies the scars attributable to tubercle, a very high proportion. It has been estimated that from 13 to 14 per cent. of the population of these islands is tuberculous, an enormous percentage. I have seen it stated recently that from 50,000 to 60,000 of the inhabitants of this country die every year from tubercle. Think of that. Use your imaginations a little. Think of a town like Reading, with its streets and its tramcars, and its public buildings, and of the people hurrying to and fro. Imagine yourself there on the first of January, 1906, and then imagine yourself there on the first of January, 1907, and not a soul of that population then alive. It is a dreadful state of things. More than that, I believe it is correct to say that more than half a million of the inhabitants of this country at the present moment have tubercle, a huge proportion. And now think of this, that they are suffering from a

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preventable disease. Of course we are all altruists, and therefore we shall make it our business to prevent this disease, and I think we ought to do so. There is something very strange about one's mental attitude towards diseases. Of course, tubercle has not been prevented because, until Koch discovered its causation, it was not known that it was caused by a bacillus. The places where the bacillus was to be found and the manner of its intrusion into the human being were not known. But they are beginning to be known. But there is something more than that behind this question. Try to consider what is your own train of thought when the name of a disease is mentioned to you. For instance, I will say that so and so has gout. You do not think much of that. Perhaps it conjures up in your imagination visions of succulent food and choice vintages, and you think possibly that the poor person who is perhaps suffering intolerable agony has earned it. Besides, gout is rather an aristocratic disease. People have no feeling of shame, I believe, in owning to the fact that they have either acquired or inherited gout. Now I will mention to you another disease, and ask you to think what that conjures up in your minds. One of the most painful things that ever happened to me in my whole life was to have to tell an officer in the Indian army that he had leprosy. Leprosy, as you are aware, is a disease which is due to a bacillus. When that bacillus finds entrance into the human body it produces there inflammatory changes, and the product of the inflammatory changes degenerates, and softens, and ulcerates, and other troubles ensue. I am sure you must all think that that is a dreadful disease. Probably the idea is instilled in your minds from the very earliest days, "a leper as white as snow," and so forth. But there is something more. The person who was supposed to be afflicted with this disease was put apart from his fellows and had to live apart, and his disease carried with it a social stigma. So obviously your notion of such a disease as that is quite different to what your notion is in regard to gout. It is a disease which carries a social stigma or stain or a social penalty with it. Now we are come to another disease, concerning which I shall lecture to-day, which to my mind is very closely akin to the one which I have just mentioned, but it is not yet thoroughly recognised to be a contagious disorder, which does not yet carry with it any social stigma, but which inevitably will when all this is recognised. Then tubercle will begin to disappear from the land, and it will be as rare in this country as leprosy is now. The leper houses of the past, the leper windows of the churches are now useless; so will be all these hospitals and sanatoria for tuberculous diseases.

Now, I have one other reason for choosing tuberculous arthritis as a subject to lecture to you upon to-day, and the reason is this—that it affords such an admirable opportunity for you to learn to apply the principles and methods of diagnosis. A tuberculous arthritis such as this boy has is almost sure to be insidious in its onset, and the patient will probably be brought to see you because he has been limping, perhaps complaining of pain in the limb, not necessarily in the joint. And now, of course, it becomes incumbent upon you to proceed with the examination of the patient. Your rules for your own conduct, as you are aware, are perfectly simple. First, as a matter of ordinary routine, you begin by examining the whole patient. Then you will examine the whole of the diseased limb; then you will compare the whole of the diseased limb with its fellow on the opposite side, taking care to carry out your examination whilst the patient is in a condition of rest. As regards the whole patient, something may be conveyed to your mind. This boy is anæmic. Look at him. Obviously the child is ill. Had it simply injured its joint, or torn a ligament, and had an effusion of blood, and gone through the reparative process, it would not have been anæmic. The child might have been in pain, but its hair would probably have been of the proper colour, its cheeks red, and probably the eyes would have been bright. And instead of its movements being languid they would have been lively and quick. In fact, we should be looking at a healthy child who was well generally, but had something the matter with his joint. When we come to look at this child, what do we see? It is said to have more than one tuberculous joint. We will take first its elbow as the joint which we will look at, and if the child has tuberculous arthritis of its elbow-joint it will have a swelling at that joint, the joint will be bent, and the muscles above and below the joint will be wasted. There is the swelling at its elbow-joint, there is the flexed elbow-joint, and there is the wasted arm. Now, let us take the flexion first. I

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think you can make a generalisation in this respect, that all inflamed joints are bent. It is easy to see in the elbow, it is very easy to see in the hip. You, of course, cannot see it in the shoulder, which lies against the side, and in the wrist you will not see it. In the hip it is easy to see if you know how. I expect everybody here has been shown over and over again how easy it is to detect the flexion of an inflamed hip-joint. All that is necessary to do, of course, is to bend the opposite limb upon the abdomen, so as to get over the compensatory lordosis or flexion of the spine, and then the bent hip is obvious. The thigh comes up from the table, for the hip may be flexed to a quarter or half a right angle. Here is a very important point. If you were to overlook this flexion in a hip, and if the chronic tuberculous inflammation of the joint were to subside, that flexion would possibly have become a permanent flexion. So permanent may that flexion have become that you might never get the limb straight by any method of extension. So operations have been devised for dividing the femur or excising its head, and oftentimes the flexion of a knee-joint has had to be overcome, not by the division of muscles, not by extension, but by actual removal of bone. So if you overlook this flexion and allow it to become permanent observe that your patient is in a very serious and awkward position. Observe, too, that the girl we are looking at has this wasting of the muscles of the arm. And as it pains her to stretch the ligaments or to put tension on the muscles, so she lifts the painful diseased arm with the opposite hand. Here you see the wasting of the muscles. I do not myself know why these muscles have wasted. It would be reasonable to say they are wasted because they have not been used, but I do not think that that can possibly be the whole truth. Because at times it occurs with such extraordinary rapidity—far quicker than any possible disuse would admit of. More than that, the wasting is, in some degree, an indication of the severity of the inflammation. In very acute septic arthritis the muscles waste with extraordinary rapidity. To your minds that wasting of the muscles should be an indication of the severity of the disease. Imagine yourself to have been called in to look at a joint like this. The mother said it was caused by an injury ; she wishes to think it was caused by an injury. Why are human beings so anxious to attribute their tumours, their bad joints, their diseases, to an injury? Surely you have thought out that problem for yourselves, and you have come to this conclusion, that when a healthy person has injured any tissue it is certain that a reparative process will take place and that it will speedily get well. That is why these people over and over again tell you that the disease began after an injury. They hope it is true because they further hope that therefore it will soon get well. But injury or no injury, the presence of the muscular wasting should put you on your guard. Now let us continue with the swelling of this elbow-joint. That swelling at the elbow-joint makes the ends of the bone look as if they were big. When the swelling is at the knee it makes the ends of the bone look very big. So it is customary to speak of the apparent enlargement of the ends of the bones in tubercular arthritis. There is another reason why the ends of the bones look big, and that is obvious too. It is because the muscles above and below the joint are wasted. Now, the first question which must invariably arise in your minds when you see a swelling in the neighbourhood of a joint is, Is that swelling inside or outside the joint?—not a particularly difficult problem to solve. A swelling outside the joint, such as she has upon the dorsum of the left wrist, obscures the structures outside the joint. So when she moves her fingers I may be unable to feel the extensor tendons. I can feel that this swelling on the back of her hand is outside her extensor tendons. So that to ascertain whether a swelling is outside the joint it is merely necessary to take some obvious land-marks—the tendons or a bony prominence—and ask yourself what relation the swelling bears to them. You generally find that the tendons are the easiest things to take. Everybody has a wrist-joint which he can easily examine for himself. Let us assume that the patient has a swelling inside the wrist-joint. Now, it is to be an inflammatory swelling inside the wrist-joint and you wish to localise it. It is inside the wrist-joint, which means it is confined to the limits of the capsule of the wrist. The capsule of the wrist-joint is attached round the head of the ulna and round the articular cartilage of the radius, which is rather higher up the arm than you would expect, perhaps, and the ramifications of the wrist-joint go down as far as the bases of the metacarpal

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bones. If you see a swelling which extends as far as the capsule of the wrist-joint goes, you cannot help suspecting that therefore it is inside the capsule. But if the swelling is inside the capsule it will not be limited to the dorsum of the wrist-joint, such as this one I have pointed out to you happens to be. The patient will also have a swelling on the radial side of the wrist-joint, a swelling on the ulnar side of the wrist-joint, and on the palmar aspect of the wrist-joint. If it is a knee swelling, the question, Is it outside or inside the knee-joint? will arise. This little girl's knee-joint swelling is within the capsule, because it is limited below by the upper edge of the tibia; above it reaches, say, two fingers' breadths above the patella. Next it is to be seen at the inside of the knee-joint, on the outside of it, and lastly, if you turn the patient over, which is a procedure that is often omitted, you will see a bulging in the popliteal space. So we are getting fairly strong evidence that this patient has a swelling inside the capsule of that joint. In the case of the knee the patella is the ordinary prominence to guide you in your determination as to whether the swelling is inside or outside the capsule. I shall now place my finger upon her healthy patella, and I at once come down upon it, and I can feel the ridges on it. I put my finger on the opposite patella and feel it with the same amount of ease. That swelling is not outside her patella. Next, having got this far, I might proceed to ascertain whether it has obscured the tendons of the inner and outer side of the popliteal space. They are rather more distinct on the diseased side than upon the other because the muscles are rather tight in the process of flexing and keep her joint rigid. Having located the swelling inside the joint, you will naturally now go to the next point with regard to this inflammatory swelling, the determination whether it is made up of something which is fluid or of something which is solid. If she has a fluid swelling inside her joint you will feel fluctuation. I might have made one further observation. When I put my finger-tip upon the patella it at once goes down on the patella, and the patella is firm upon the condyle of the femur. Had it been floating upon the surface of a fluid effusion I should have felt it give under my fingers, and should have felt it go down with a tap upon the condyle of the femur. That, to my mind, is rather a rough test. So you ought to proceed to apply the ordinary test, and see whether you can pass a wave through the joint from side to side. Here is a swelling at the inner fossa of the knee and at the outer, and when you try to make a wave from side to side and try to get fluctuation you cannot. I am confronted therefore with a solid swelling, which, of course, is incapable of fluctuation. That solid swelling inside the capsule of the knee-joint has a few other characters. For instance, had this been fluid in the joint, obviously there would not have been very much of it, because I can still see traces of the fossæ. And that fluid would not be particularly tight. So when I press with the tip of my finger I ought to come down at once through the fluid upon the external condyle of the femur and upon the internal condyle, upon the edge of the trochlear surface of the femur, upon the sharp edge of the inner condyle of the femur. On the left side, which is presumably healthy, I can make out the bony points which I have spoken about, but on the diseased side it is very difficult to feel either the internal or the external condyle. In other words, they are obscured by this solid swelling inside the capsule of the knee-joint.

Now, it is important to you to be clear as to the course which this inflammatory swelling inside the capsule of the knee-joint will pursue. Tubercle in any part of the human body pursues a constant course; but when I say that, you must not assume that every possible case of tubercle will run this course from beginning to end. But it very likely will, and you ought to be prepared in your minds for it running this course. The first step in the onset of tubercle is, of course, that it should be implanted in the human body. I suppose that for our purposes we may take it that it passes through the respiratory tract or some part of the alimentary tract. Or it may be implanted upon the surface of the body. It is not particularly rare to meet with people who at some period or another of their lives have cut themselves and become inoculated with tubercle. When that is the case it is not infrequently mistaken for other diseases, and especially one in which the unfortunate victim is condemned to a very long course of mercury. Supposing that the tubercle bacillus has got into the body through the tonsil, we will say, it then is carried from the lymphoid tissue of the tonsil into the lymphatics of the neck, and it is easy for you, in the case of the lymphatics of the neck, to trace

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step by step the course which this disease is pursuing. The first effect of the tubercle bacillus is to inflame the lymphatic glands in which it is lodged. The first effect upon the knee of this girl, and upon her shoulder and her wrist, is an inflammation of the synovial membrane in which it is lodged. Whether she has tubercle in the cancellous tissue of the bone in proximity to this joint I do not know, but I do know for certain that she has thickening of the synovial membrane. This inflammatory condition of the gland or of the joint makes either of them hot and slightly tender. The swelling within the capsule is rather firm, but can be indented with the finger. But soon a change takes place in the tuberculous inflammatory tissue, and it undergoes a change which is called "caseation." Here is a museum specimen of a tuberculous gland in which the inflammatory product produced by the tubercle bacillus has undergone this change, which is caseation. I shall not talk to you now about the histology of this condition, because obviously the first step for you gentlemen is to learn to use your naked eye, and to learn what you can accurately see with your naked eye. When you are sufficiently versed in things which you can see with your naked eye you can proceed to use the more fallacious and misleading kinds of investigation, namely by means of microscopes. You can see in the gland those caseous lumps. I wonder why the inflammatory products become altered into that caseous material. I have asked Dr. Andrewes if he would lend me a culture-tube of tubercle—tubercle growing on glycerine agar. And I think that anyone glancing at that tubercle growing from a minute implantation which originally could not be seen would say that there is the very closest resemblance in colour and consistence between the tubercle growing in that test-tube and the caseous material in the lymphatic glands. I ought to tell you that I have often looked at the tuberculous material which can be procured from a test-tube such as that. And stained on a cover-glass, it is an extraordinary mass of tubercle bacilli. If you are expecting to see the same appearances in a scrap of the caseous material removed from that gland you will be disappointed. I question whether you would see any bacilli at all unless you were to spend hours in the attempt. I expect you would see a mass of granules, and the field of the microscope, if it were a smear preparation, would look as if it were rather obscure ground glass. I do not know, but I always think myself that the tubercle bacilli in forming that caseous material must have undergone a process of degeneration, and that it contains, not tubercle bacilli, but the products of degeneration of the tubercle bacilli. At all events, whatever it contains, it is an infective material which when passed into the lymphatics or into the general circulation of a human being or an animal will cause tubercle. Now, here is a specimen showing tubercle of the kidney. And that shows the next alteration which takes place in that caseous material. Caseous material in a gland makes that gland feel extraordinarily hard, so that you are unable to imprint it with your fingers. And more than that, the gland has probably ceased to be tender or painful. But soon an alteration takes place in the gland, and you can see it taking place in this specimen in the kidney. Each of those tuberculous lumps in the kidney is hollow; the centre of each tuberculous lump has become soft, it has become filled with pus, with softened material which I shall call tuberculous pus. I have no doubt that this tuberculous pus may have been washed out in the process of putting up the specimen, or it may have run down the kidney tubules into the urine. But this has been the course of events: tubercle bacillus, inflammatory products, caseation of inflammatory products, softening of inflammatory products. Observe the clinical phases which correspond to that. When the tubercle bacillus enters the tissues you do not know it. When it has got into the tissue and begun to inflame you will be aware of it, the structure takes on the ordinary characters of inflammation. When the tuberculous products have caseated, the diseased parts are hard and not at all tender. If the caseous product be inside the capsule of the lymphatic gland it feels like a bullet, and it slips about in the neck. Now a change takes place, and for some reason the caseous material softens, the gland becomes soft, and now it fluctuates, and you say that the patient has a tuberculous abscess. Now these tuberculous abscesses in any region of the body proceed to make their way to the surface of the body. The method in which they make their way to the surface is quite a simple one. So far I have been speaking of this tuberculous process as being confined within the capsule of the gland or of the

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joint. Soon the inflammatory process passes to the capsule, and then beyond the capsule. Then the tissue outside the capsule of the gland is inflamed too. The kind of inflammation which they are suffering from is, of course, tuberculous inflammation. How shall you tell clinically when the inflammatory process has passed from the interior to the exterior of the capsule of the gland, onwards to the cervical fascia, to the platysma myoides, and to the skin? It is simply that the inflammation has passed through the capsule, which has become adherent to its surroundings. The tuberculous inflammation proceeding in the deep fascia of the neck, and platysma myoides, and skin of the neck, undergoes the same process. It may caseate, it often does, it is sure to soften, and the result is a tuberculous ulcer. A probe passed into the centre of that tuberculous ulcer passes into a tuberculous sinus, which leads you into the centre of the tuberculous gland. You may observe that in this child the same process has been going on in the carpus. I have no doubt she has had here also tuberculous disease, and the tuberculous inflammation is proceeding at the present moment. The tuberculous material has caseated, it has softened, the softened material or pus has escaped, and then, whether by art or Nature, she has managed to heal this abscess cavity and sinus. Supposing the tuberculous disease in her other joints is not arrested by Nature or by art, what will its progress be? The progress will be the same in her knee and her elbow as it would have been in one of the tuberculous glands in the neck. First, it may be tuberculous inflammation of the synovial membrane inside the joint. Then that membrane at some point will caseate and then the caseous material will soften, and then the pus might be discharged inside the joint. Then there will be an extensive tuberculous disease of the synovial membrane lining the joint. Or it might spread to the capsule of the joint and to the tissues covering the joint. Then it softens and an abscess may be opened, and the tuberculous pus escapes. But think of a tuberculous sinus passing from the inside to the outside of a joint, its passage through the septic skin, its exposure to the open air, and then you can easily imagine the joint becoming septic by way of the sinus which has been allowed to form or been created. Now imagine to yourself the course of an acute septic arthritis of a joint implanted upon tuberculous arthritis. I can easily believe that a gentleman who has not carefully thought as to whether an abscess which is forming at the inside of the knee-joint and which he had guessed was an abscess outside the joint, and had opened on that assumption, would be rather puzzled by the attack of acute septic arthritis which might be reasonably expected to supervene upon the opening of that abscess if it were not done with the most accurate aseptic precautions.

I have only just touched upon the fringe of a very great subject. You may observe I spoke of tuberculous arthritis, but you cannot speak of tuberculous arthritis apart from tubercle of any part of the human body. Next time I will continue and speak of the course which tuberculous arthritis pursues, and will endeavour to explain to you the methods of treatment which are adopted for it and why they are adopted—in other words, the reasons which ought to exist in your mind for the course of treatment which you adopt.

July 9th, 1906.

A LECTURE

ON

THE NECESSITY FOR AND THE NATURE OF THE TREATMENT OF BACKWARD DISPLACEMENTS OF THE UTERUS.*

By THOS. G. STEVENS, M.D., B.S.Lond., F.R.C.S.Eng., M.R.C.P.Lond.,

Obstetric Tutor St. Mary's Hospital; Assistant Physician Hospital for Women, Soho Square.

GENTLEMEN,—I will make no apology for addressing you to-day on so apparently a trivial subject as the treatment of backward displacement of the uterus. It is so common a condition that all of us are called upon constantly to deal with it, and yet there are few complaints in the category of gynæcological diseases which give the subjects of it and *us* so much trouble. They do not cause us anxiety in the same way that laparotomies do, but they form the bulk of our practice and are often much more difficult to treat than well-declared conditions, such as tumours of the pelvic organs.

* Delivered at the Medical Graduates' College and Polyclinic.

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The treatment of pelvic tumours is well defined, and can be carried out by any one who has surgical skill and a knowledge of technique, but to treat successfully a case of retroversion and flexion requires careful investigation, accurate interpretation of symptoms, and then a decision as to whether special treatment is required or not. The latter part of the question is the crux of the whole subject, for there can be no doubt that it is so simple to put in a pessary that this almost necessarily follows in most cases. And yet, to the great disgust of patient and practitioner, the putting in of a pessary fails in many cases to relieve the patient's symptoms, and then follows a time of tribulation for both. We are all familiar with this sort of case, and for some time past now I have set myself to try and avoid these unpleasant experiences by making a more careful interpretation of the patient's symptoms so as to decide whether local treatment will relieve them or not. Of this I am sure, that a pessary which relieves no symptoms had far better never have been applied. I might well have taken for a text to-day the query "Will a pessary relieve this or that patient's symptoms?" We all have a feeling in the presence of a retroversion and flexion that we must do something to it, and there is nothing so easy to do as to put in a pessary.

After some years of out-patients' work in a women's hospital, I am now quite convinced that a large number of backward displacements require no special treatment at all, and the reason for this, I am sure, is because such displacements often cause no symptoms. The patients have symptoms and have a displacement, but they stand in no relation to one another.

In order to have a clear understanding of these cases we must have some conception as to their causation, for upon this will often turn the question of interpretation of symptoms. There are two great classes of backward displacements, namely the congenital and the acquired. Although mentioned in text-books, the former cases have not received the attention they merit, and to this fact must be attributed much of the ill success in treating them. They are very common cases and are often discovered when examining patients who complain of symptoms possibly attributable to the pelvic organs. On the other hand, there is no doubt that acquired backward displacements form a class larger than congenital cases, but they occur in married women, and sometimes have much more definite symptoms associated with them. It does not follow, however, that a backward displacement in a married woman requires local treatment and in an unmarried one does not. This is far from being the case. The condition may exist in both classes and yet require no treatment, according to the symptoms being caused by the displacement or not. For all this it is a fact that backward displacements in married women much more often require local treatment than in the unmarried. The reason for this depends on the causation, for by a clear idea of this we can arrive at the reason why some displacements cause symptoms and others do not.

The gist of the matter is this: those backward displacements of the uterus in which that organ has been pregnant or has been infected are liable to remain congested for a long time, and as a result may get endometritis or overgrowth of the components of the endometrium. It is this congestion or endometritis which really causes the symptoms properly considered to belong to backward displacements. The converse follows, that a backward displacement of a uterus which has not been pregnant or infected is not usually congested, and therefore does not cause symptoms.

The congenital retroverted uterus is not congested because there has never been any cause for congestion in most instances. If, however, a congenital retroverted uterus becomes pregnant or infected it may cause symptoms, and then the case becomes indistinguishable from an acquired one.

The causation of an acquired backward displacement is nearly always parturition or abortion, and I had almost said that there is no other cause. This is perhaps going a little too far, but what may be said is this, that a uterus in a healthy individual with healthy adnexa practically cannot become displaced except as a result of pregnancy. In a healthy person the uterus cannot be displaced permanently by an accident. Such a cause is often attributed to these cases, such trivial accidents as over-reaching, falls downstairs, from riding a bicycle, etc., are the common histories one gets. When we consider the nature of the healthy uterus, its attachments and position (see Fig. 1), we cannot but feel that these accidents cannot possibly cause permanent backward displacement. On the other

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hand, when we find a backward displacement and the history of an accident given, we almost invariably find that the displacement is congenital. It is just possible that an unhealthy person living in unhygienic surroundings with insufficient or improper food, may as a result of relaxed connective tissues and habitual over-distension of the bladder acquire a backward displacement. The over-distension of the bladder is necessary, for some actual mechanical displacing cause must be present, the other accompaniments only helping to render the displacement permanent.

not forget that retroversion may itself be a cause of subinvolution.

The infected but non-pregnant uterus may become retroverted in the same manner, for all its ligaments share in the softening effect of inflammation, and being heavy in addition, it may drop backwards to be kept there by intestinal pressure. Afterwards such a uterus may become fixed by adhesions if there is any pelvic peritonitis.

These two, then, are the prime causes of acquired backward displacements, and practically speaking there are no others, and with these two are associated

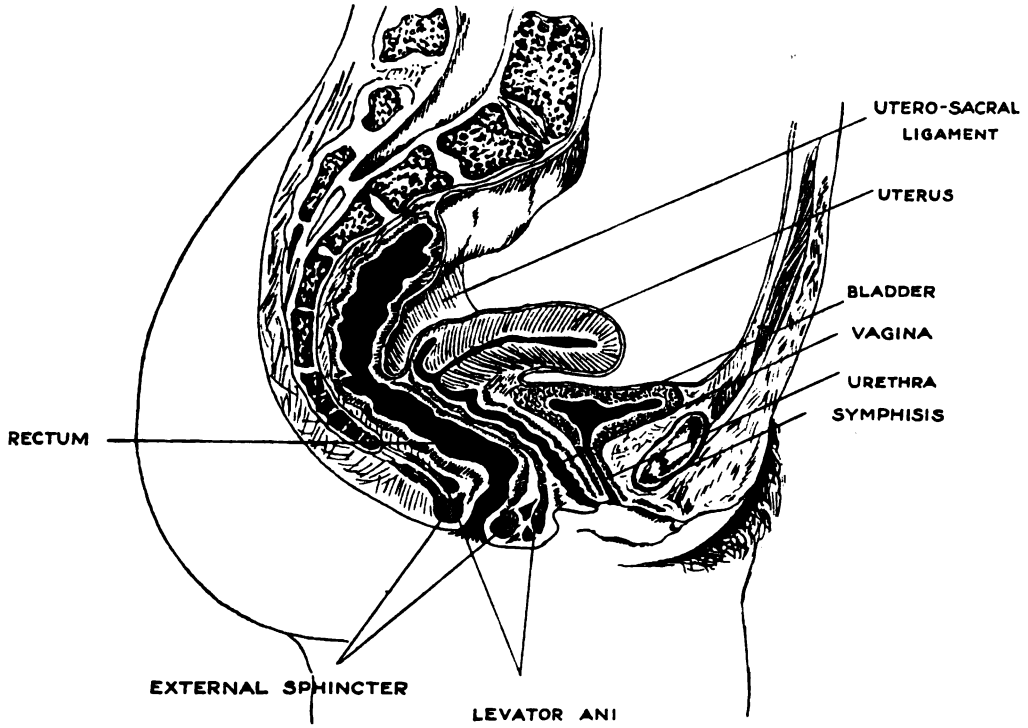

FIG. 1.—Illustrates the normal relations of the unimpregnated uterus (after Dickinson).

Parturition causes a backward displacement because it is the custom in this country for women after labours to lie upon their backs. The effect of this may be that the heavy uterus gradually falls back until the fundus gets small enough to descend below the promontory of the sacrum, and then the intestinal pressure getting on its anterior surface keeps it down in the retroverted position. When such a patient gets up it is a mere chance whether the uterus assumes its normal position or remains retroverted. This acts more potentially if there is any subinvolution, although we must

the causes of the subsequent congestion to which the real symptoms of this condition are attributable.

It follows from the above-mentioned causation that acquired backward displacements are always associated with enlargement of the uterus (compare Figs. 2 and 3, which show different stages of acquired retroversion with Fig. 4, which depicts a congenital case), and if causing symptoms, with uterine congestion. This congestion may affect also the ovaries and tubes, and of course their attachments. As a result we often find the so-called prolapsed ovaries in these cases. This is

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really descent of the ovaries from their normal position until one or both rest somewhere on the peritoneum of either the lateral cavities of the pelvis or of Douglas's pouch. Even if the uterus shows no symptoms of congestion the prolapsed ovaries may be painful (*i. e.* congested), because there can be no doubt that the folding of the broad ligament accompanying retroflexion interferes with the normal venous return from the ovaries.

The symptoms that a retroverted and retroflexed congested uterus can give rise to are: enlargement of uterus; backache; tender ovaries; increased mucous discharge, leucorrhœa; menorrhagia and dysmenorrhœa; tendency to abortion, sometimes repeated abortion.

All these, we must agree, are due to congestion, which we have seen is secondary to or accompanies the displacement.

On the other hand, if we examine a case of congenital backward displacement we find a very different condition. The uterus is nearly always smaller than usual, this I have attempted to show in Fig. 4—a very important point, for it signifies mal-development of the organ, in other words, a congenital malformation along with a congenital malposition. The ovaries are very rarely found to be prolapsed, generally, I believe, because the broad ligaments are short and the ovarian attachments not elongated. If the ovaries are unusually tangible, they are not tender to the touch. (Note, unusual tangibility of the ovaries signifies either descent, or hardening the result of chronic inflammation.) The vagina in these cases is very short, the posterior wall markedly so, and more nearly approaches the anterior in length. This condition is illustrated also in Fig. 4, which may be compared with Figs. 1–3, which show the relations of the parts in normal uteri and acquired displacements.

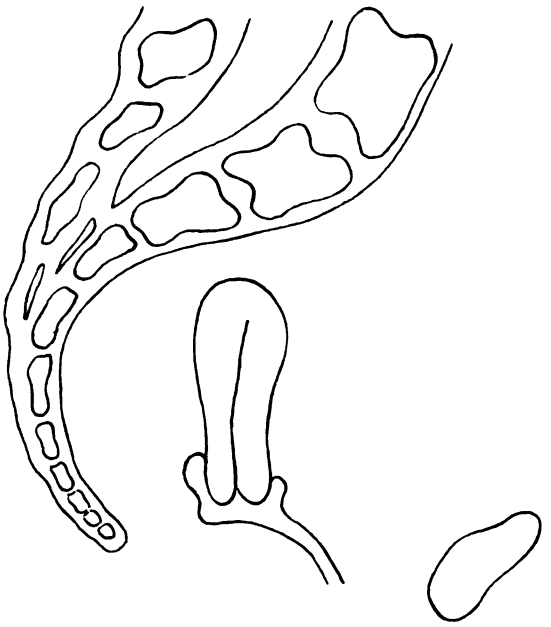

FIG. 2.—The uterus retroverted until it has assumed a vertical position.

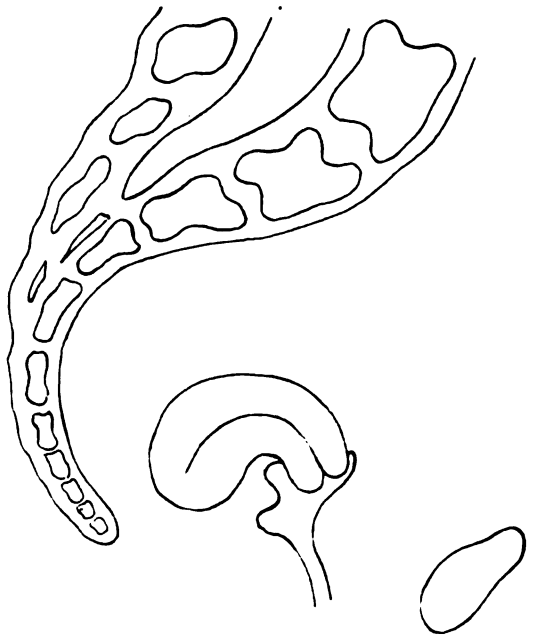

FIG. 3.—The completely retroverted and retroflexed uterus (acquired).

Such a uterus gives rise to no symptoms peculiar to itself, except that there is often some anomaly of menstruation. This, however, is not menorrhagia, but more often scanty and painful menstruation—in fact, the kind of menstrual disorder associated with a congenitally under-developed uterus, and having nothing to do with the displacement.

Like acquired retroversions, these uteri do not seem to be debarred from conception—in fact, some subjects of them conceive readily. This, however, depends on the degree of mal-development, and in any case I believe that this form of uterus is not nearly so often associated with sterility as the small acutely anteflexed uterus so

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often associated with spasmodic dysmenorrhœa. An interesting point with regard to conception in these congenital cases is that if undiagnosed and left alone abortion nearly always occurs. If, however, the pregnancy is diagnosed within the first ten weeks it is quite easy to replace the uterus in an anteverted position and keep it there with a pessary. Replacement when pregnancy occurs is possible, because the vaginal walls and ligaments become relaxed, allowing the uterus to be retained in an anteverted position. I have been fortunate enough to see several cases go to full term after this treatment. The pregnancy seems to lead to

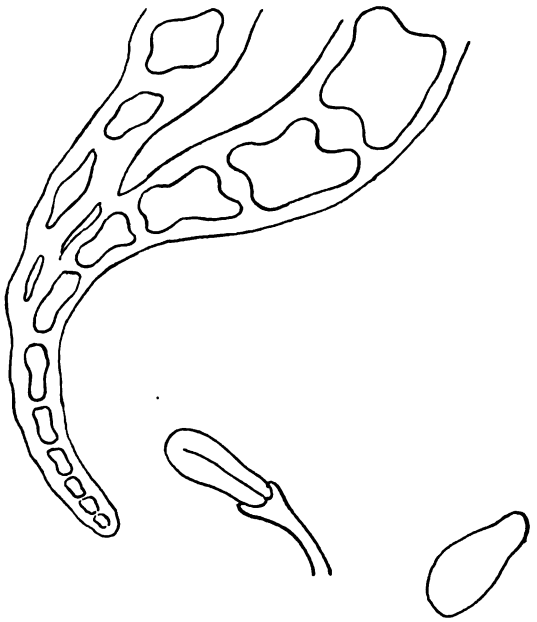

FIG. 4.—The congenital retroverted uterus.

development of the uterine muscle in a way that nothing else will. The retroversion, however, recurs invariably, I believe, after labour.

The subjects of these congenitally displaced uteri often come before us with symptoms, some of which may easily be confounded with those really associated with displacements. The commonest symptoms are backache, constipation, dyspepsia, scanty and painful menstruation, and dyspnœa, the result of anæmia.

With regard to these symptoms backache is, perhaps, the most difficult to explain. It occurs in all kinds of uterine conditions, with or without displacements and with or without congestion; therefore, it is impossible to say to what extent the displacement may be answerable for it. But a careful examination in many instances will show that it is due to something else altogether, one of the commonest being some change in the urinary constituents such as lithiasis, phosphaturia, or hyperacidity. In others an examination of the superficial nerves will show that it is associated with a neuralgia, sometimes intercostal, sometimes lower down, but generally revealed by tenderness on deep pressure over the nerve where it issues from the spinal canal. Sometimes it is due to a lateral curvature, the result of a faulty position of rest. In other cases if high up it may be a manifestation of dyspepsia, or when low down even caused solely by constipation. I have said enough here at any rate to show that backache is not by any means necessarily caused by a backwardly displaced uterus.

With regard to constipation, let us at once agree that backward displacements do not cause constipation, nor do they have any effect upon the rectum at all. One of the common beliefs I often hear is that retroversion causes hæmorrhoids. I am quite sure it does not. Constipation and hæmorrhoids are so common in women both who have and who have not backward displacements of the uterus that there cannot be any causal relation between them. The ordinary forms of backward displacement are so movable that they cannot and do not exert any pressure on the rectum or its vessels.

The same can be said at once of dyspepsia and anæmia; they have nothing to do with backward displacements of the uterus.

I have already pointed out that the scanty and painful menstruation is due rather to the congenital maldevelopment than the position of the uterus.

If malposition does anything to menstruation it eventually causes menorrhagia from congestion.

From these statements, then, it is clear that as far as symptoms go congenital backward displacements do not in the majority of cases have anything to do with their causation.

For these reasons I believe that these particular cases require no local treatment, and are best left alone.

Supposing local treatment is tried in such cases—and usually this is the insertion of a pessary—what do we find? First of all we find invariably that the displacement is not corrected, even if we know

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that the uterus had been anteverted before insertion of the pessary. The reason for this, I have no doubt, lies in the short posterior vaginal wall. For in trying to antevert these uteri one can feel this wall gradually getting more and more tense, and on leaving hold of the uterus the fundus is dragged back at once into its old malposition by the resiliency of the short posterior vaginal wall. A pessary makes no difference to this; in fact, it makes the condition worse, for the more tense we make the posterior vaginal wall in these cases the more traction it exerts on the cervix and so retroverts the uterus. As I have shown, the condition is very different when pregnancy occurs. A uterus can only be kept in position by a pessary when the posterior vaginal fornix is roomy; there is practically no posterior fornix in these cases.

Next we find that the pessary has not relieved the symptoms. This is the worst feature of these cases, for if a pessary does not relieve symptoms it is worse than useless, for we are all familiar with the inflammatory vaginal conditions started by pessaries even when they are doing good in other directions.

In my out-patient work I often remove pessaries put in by other people. Sometimes this is followed by relief, and I cannot remember a case in which any increase of suffering has resulted.

Another point always appeals to me, and it is this: the insertion of a pessary for a young unmarried girl does much harm psychologically, by constantly calling attention to the genital organs, which often have nothing to do with the symptoms complained of.

The successful treatment of these cases depends on a correct interpretation of the symptoms. Whatever we judge after a careful examination to be the cause of the symptoms we must treat in its own appropriate manner. Dyspepsia, urinary deposits, neuralgias, anæmia, constipation—all of these can be successfully treated, and it is surprising how simple internal medication will relieve many of these patients. And, not least in such cases, it is much better that the patient should not be told that she has a displacement; she is only too likely to think about it, and having heard of pessaries to expect to get one from somebody. I have seen such cases, and in the end they have been bitterly disappointed because the pessary did more harm than good.

Turning to the acquired backward displacements, as I have already pointed out, if they cause symptoms referable to the displacement, these are really due to congestion and its secondary result endometritis. I will not enter into the vexed question as to the cause of this congestion; no doubt it is sometimes solely the result of interference with the blood-supply, whilst at others it is due to infection. The result is much the same, endometritis, characterised by overgrowth and thickening of the endometrium.

Many of these retroversions and flexions, although acquired, have no congestion, and do not, therefore, cause any symptoms peculiar to themselves. They therefore require no special local treatment, and may be considered in the same category as the congenital cases.

In the congestion cases, however, two symptoms stand out pre-eminently, namely menorrhagia and tender prolapsed ovaries. These are the symptoms which more particularly call for local treatment. Whatever this treatment is, to be successful, these two symptoms must be relieved and the others will certainly improve—I mean the backache, leucorrhœa, etc. It is not possible to forecast whether replacement and pessary treatment will relieve these symptoms. The probability is that pessaries alone will not cure cases which are infected, but will probably give much relief in cases where the congestion is solely due to the displacement. So, then, in these cases pessaries may be tried. If the ovaries are not prolapsed and tender, replacement may be performed at once and a suitable pessary put in. With regard to replacement, let me warn you against the use of the sound for this purpose. It is unnecessary, painful, and dangerous. Unless all the generative canal has been most carefully made aseptic there is always a risk of carrying infected material on the sound up towards the fundus, and I am convinced that many cases of endometritis and tubal inflammation have been started this way. If it is not possible to replace the uterus bimanually with the fingers alone, the best course to pursue is to fix the cervix with a tenaculum in its anterior lip, draw the uterus down, and then push the fundus forward with the finger in the vagina (Fig. 5). This manœuvre never fails, except where there are adhesions between the uterus and Douglas's pouch. It is devoid of danger, and patients seem to feel very little pain from the prick of the tenaculum—much less, in fact, than by prolonged attempts at replacement with the fingers. We cannot by this means alter a retroflexion, but we can antevert the uterus as a whole in its retroflexed position. It is said that the fundus may be gripped and encarcerated by the utero-sacral ligaments; I can only say that I have never met a case in which this could be proved to be so. The downward intestinal pressure on the anterior surface of the uterus is

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the real bar to replacement in these cases. When adhesions exist there is nearly always a history of pelvic peritonitis, and evidence of old salpingo-oophoritis, not difficult to diagnose as a rule. The pessary used does not matter very much; those in common use—namely, the indiarubber ring and the vulcanite Hodge—answer equally well. Where the vaginal outlet is narrow the Hodge is, perhaps, better; with relaxed outlets the ring will keep in the vagina better. The one object, however, should be to put in the smallest pessary that will stay in, and that will keep the uterus in position. Too often too large a pessary is chosen, which, of course, is difficult to put in and take out, which over-stretches the vaginal walls, causing pain, and,

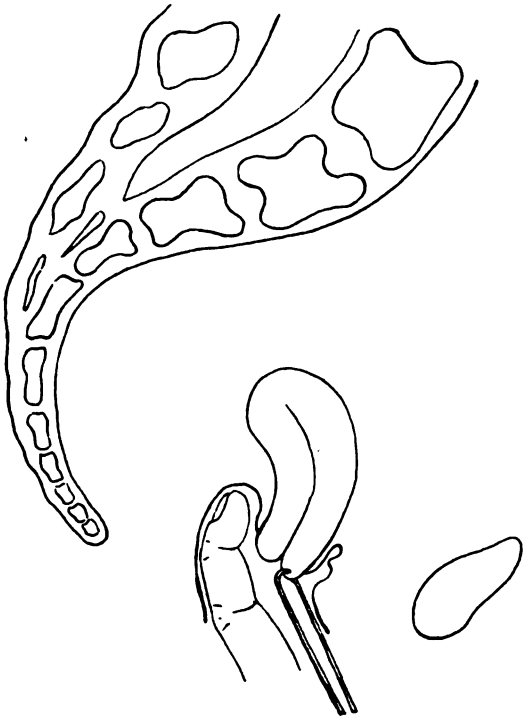

FIG. 5.—Method of replacement with the vulsellum.

worst of all, setting up vaginitis from irritation and infection.

If the ovaries are prolapsed and tender, it does not do to put in a pessary at once. The congestion must be first relieved, and there is no better way of doing this than by morning and evening copious hot douches, with an ichthyol vaginal suppository every other night. The suppository is made of 10 per cent. of ichthyol in 120 grains of cacao butter. This is put in after douching and its remains washed out next morning. It acts by causing a copious flow of mucus from the cervix, so relieving congestion. It sometimes coagulates the superficial vaginal epithelium and brings away a cast, but this does not matter. At the same time, medicinal treatment must be carried out; ergot in large doses (3j of the liquid extract) must be given internally thrice daily, and constipation carefully treated.

If this treatment fails to relieve menorrhagia and backache, the uterus is probably infected, and will have to be thoroughly curetted after dilatation, replacement being left until after recovery from this. There still remains a proportion of cases in which the treatment does no good. The uterus remains retroverted in spite of pessaries, or the ovaries remain prolapsed and tender, producing the worst kind of dispareunia, and along with these the cases with adhesions must be considered. Pessaries, of course, are useless for both, and the question of radical operative treatment must be considered.

This will come under the heading of Fixation Operations, or Shortening the Round Ligaments.

The latter has not received the amount of support that, perhaps, it merits. It is only really suitable to cases without adhesions, although some American operators have gone so far as to separate adhesions through the opened up inguinal canal. This is so altering the character of the original operation as to make it as serious or more serious a procedure than ventrofixation. The particular virtue of the original operation lay in the fact that the peritoneum was not opened, and that there was no tendency to inguinal hernia afterwards. There is no doubt that Alexander's operation is physiologically ideal; but when we consider that sometimes the round ligaments cannot be found, or that when found they may be so slender as to be incapable of bearing any strain, two at least of its limitations are made manifest. On the whole, the balance of opinion in this country is in favour of a fixation operation, and most operators favour abdominal or ventro-fixation. The chief argument against these operations is that they cause dystocia if a subsequent pregnancy occurs, quite apart from any risk attaching to the operations themselves. There is no doubt that ventrofixation is much easier to perform than vaginal fixation, and from the published cases the proportion of difficult labours afterwards seems about equal in each. The principle on which these operations should be performed is to fix the fundus *only* to peritoneum; in this way subsequent difficulties in labour can be avoided. By this means a stretchable band of peritoneum forms between peritoneum and fundus, allowing the uterus to rise if pregnancy should occur. To enter fully into the details of these operations is beyond the scope of this lecture, but this much we can safely say: these operations are quite justifiable under the conditions named, they are so safe that they should have no mortality, the results are quite good enough to justify them also, and ventro-fixation is the one which will be found to be favoured by most operators.

July 9th, 1906.

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NOTES FROM PRACTICE.*

By W. J. TYSON, M.D., F.R.C.P., F.R.C.S.,
Hon. Physician, Victoria Hospital, Folkestone.

I HAVE been thinking how much experience and "out of the way" knowledge we must possess after some twenty or more years of practice, and yet how seldom we commit this experience to paper. In many men this knowledge remains, so to speak, in solution; every now and then it may be crystallised and made of definite use, but much of it is lost except to the possessor, never to be regained. Perhaps one of the main reasons of this is that when a man has gathered much he is too busy to record it, and so it comes to pass that when men have little to do and less experience they write books, and when they should write books they have no time. This evening I propose to run over lightly some half a dozen subjects which appear to me to be of interest.

Prognosis in Heart Disease.

When I was a student the mitral and aortic diseases made up most of the diseases of the heart, and any one with a mitral bruit or aortic bruit was looked upon as in a bad way. On entering practice after giving bad prognoses in some cases of aortic and mitral disease, one found oneself often in error, the patient living on and in many cases in good health and working power in spite of evil forebodings. I have kept a record of cases with well-marked valvular disease; and although this record was commenced twenty or more years ago, some of the cases are alive and well to-day.

You may remember that many years back a "Collective Investigation Committee" was appointed, which did very valuable and serviceable work in collecting knowledge about the history of various diseases. I suggested to that committee that the subject of prognosis of heart disease should be taken up; they agreed with me, and Sir Andrew Clark read the opening address at the Annual Meeting of the British Medical Association at Brighton on this subject. His tables included 500 cases of cardiac disease, with their histories.

But what I now particularly wish to draw attention to is, not the valves of the heart, but the wall, which is in my opinion of more importance. One has learned from epidemics of influenza, from diphtheria, rheumatism, gout, syphilis, and the many toxic forms of poisoning, that the wall of the heart is more apt to give out than the valves. Or again, that excessive breathlessness which follows exertion in those who are untrained came out with great prominence in many of those who took part in the late Boer War. I had the opportunity of examining some of these cases at Sandgate. The mitral bruit seems to be produced by the dilatation of the left ventricle, which latter was very marked in these cases. The bruit in many of these over-strained hearts entirely disappeared and the left ventricle regained its natural size when proper rest was maintained. One must not forget that hospital heart cases, which are the only ones that we see as students, are generally of a serious character, and occur among those people whose environment and habits have not been such as to insure a healthy cardiac muscle, and we enter practice with very often quite wrong views as regards prognosis of heart disease. The subject is too wide for me to enter farther upon it, but there is one point, an ethical one, which I particularly wish to draw attention to, and that is the common practice of telling people that they have a weak heart. I know people who have been practically ruined and rendered useless for life by being told that they have valvular disease. The less one thinks about one's heart as a rule the better. A careful and experienced doctor, with the aid of the patient, may find out fairly accurately what the heart in question is capable of doing, and then some common-sense rules can be laid down. Dr. Goodhart said the other day, in concluding his Cavendish Lecture on diseases of the heart, before the members of the West London Medico-Chirurgical Society: "I am sure of this: we do far more good in our day by pronouncing people sound, if out of health, and by sending them forth with strengthened hope, than by keeping them in the chains of uncertainty and nervous dread, than which nothing can be more crushing to the utility of a life or more harmful to the chance of regaining health."

On Albuminuria.

The ordinary text-book's classification of Bright's

* A paper read before the Ashford Division of the British Medical Association.

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disease, viz. acute Bright's disease, such as that which follows cold or scarlet fever ; chronic tubular nephritis or smooth white kidney, and interstitial nephritis or the granular and contracted kidney, does not by any means cover our cases of albuminuria which we meet with in private practice.

I am not referring to those cases of cyclic or functional albuminuria which have often been described and which we know so well, nor to those cases which follow temporarily specific fevers, nor to those associated with chronic congestive states of chest and abdominal organs. Cases that I am now referring to occur in people who are for the most part pretty well, the albumen is almost constantly present, but not as a rule in large quantity. Let me give you an example in point. C. B—, now æt. 55 years, when twenty years old was refused admission to life assurance by a well-known London physician on account of the presence of albumen in the urine. Some ten years back I was consulted by the same person to consider again the possibility of his life being accepted for insurance. I found a marked quantity of albumen, but considering his past history, advised the insurance company to accept him on the principle of heavy premiums to be paid at the beginning, and to be gradually but steadily decreased as life went on ; and should he live the usual appointed years, the amount paid in all should not amount to more than the total of ordinary premiums. My friend is alive to-day and fairly well. Now, although this is no doubt an extreme and exceptional case, yet there are many cases of albuminuria which come under none of the recognised classes of Bright's disease. I believe that there is a fair number of distinctive clinical divisions of albuminuria, not mentioned in our ordinary books. Whether they should be definitely classed under the term " Bright's disease " may be doubtful. If the presence of casts constitutes organic disease, then they might be, for casts are certainly sometimes present. Because you find albumen in the urine do not be in any hurry to class his case under one of the classical divisions of the disease. I am convinced that many live for years without ever having a granular kidney, certainly not a large white one. Whether some of these cases may not come under the head of what is called the large red kidney I am not sure.

On the Soporific Action of Mercury.

I am not aware that the above action of mercury in the class of case I am about to describe is at all generally known, and that is my reason for bringing the subject before you. The cases in which I have found blue pill—for this is the form of mercury I am referring to—give such good soporific effects are rather difficult to describe and must be given in a more or less general way. Many men would call them cases of biliousness, and for want of a better term, I am contented to use it provisionally. The patients are usually over forty, complaining of lassitude, loss of appetite, a general fulness of the abdomen, pains in the shoulder-joints, tongue generally of a whitish-brown colour, a nasty taste in the mouth, eyes are rather " thick," a want of clearness of thought, more or less dull pain in the head, not confined to one particular spot, irritability of the skin, and above all *sleeplessness* at night ; there may be many more symptoms and signs than the above, or few of them may be present, but when the symptom of sleeplessness is prominently complained of, it is then that we find the soporific action of blue pill followed by saline draughts peculiarly brought out. The symptoms I have detailed are principally those found under the heading of " bilious dyspepsia," but there is this important clinical difference : whereas sex, richness of food, and want of exercise play leading parts in producing this particular form of dyspepsia, the cases that I have in mind are found as often, if not more so, in women as in men, and where the plainest diet and fair exercise have been taken. There is little doubt that sea air has something to do with this state of health ; people who have come down to the seaside after a long residence inland frequently develop after a few days all the symptoms described. I believe that we who practise in seaside resorts would less often be called upon to prescribe for this condition were a little mercury taken by the patient immediately on his arrival.

I do not associate these cases with those which are commonly called " lithæmia," or the substitution of uric acid for urea as the final product of disintegration of albuminous substances within the body ; in this last condition you obtain the more remote symptoms of gout.

What is the exact action of the mercury which

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brings about the happy result? I do not know, nor am I anxious to speculate or propose theories. Murchison supposed that mercury possessed a double action, for he says whether or not the secretion of the bile was increased, there was certainly more bile passed when the drug was being taken, and an eliminating action was brought about, so that less of the bile constituents were absorbed from the intestine than usually. Again, Murchison supposes in some way or other the albumen is more thoroughly disintegrated. In some such way the brain is "cleared." It is important for us to know empirically the use of a drug which will enable one to give a patient a good night's rest whilst at the same time you are treating the origin of the trouble and apparently not giving ordinary soporific drugs.

The Danger of putting Old People to Bed.

Probably I shall be running atilt against someone's opinion when I say I believe that many old people are sent to bed too early, and are kept too long in bed when they are convalescing. Of course there are many obvious exceptions which will at once occur to you, and which I need not take up your time in mentioning. I believe that we should be chary in advising old people to take to their beds when slightly ill. Some few years ago I was attending an old man of eighty with a slight attack of bronchitis, the like of which he had often suffered with before. A consultation was suggested, and the consultant called in advised my patient going to bed. I remonstrated, but gave way to what I thought then was wiser advice than my own. The patient kept his bed for a week, and then died. It may have been a case of *post hoc* and not *propter hoc*; still, my experience has since led me to believe that this man would have had a better chance of recovery if he had remained up during the daytime.

We must remember that the will power of old people is comparatively weak, and that bed to them is comfortable, then when once in bed after a few days the heart and muscular system lose tone. The effort to overcome this is difficult and sometimes impossible to obtain again. It often requires a good deal of courage to keep these old people from taking to their beds, for the patient often takes kindly to bed himself, and the friends think you unkind in advising less rest. My own belief, then, is that it is wise to keep old people about and with moderate exercise as long as possible.

On Cases simulating Typhoid Fever.

For some years past there have come under my notice cases which have often given me trouble in diagnosis. The patients affected are of all ages, and of both sexes. The common symptoms have been a general malaise; abdominal pain of a rather general character, but having a tendency to concentrate itself around the stomach area; a furred tongue; temperature varying between 101° and 103°, not necessarily higher in the evening, although often so; pulse of good quality. Although there is no desire to take food, yet sickness or nausea is generally absent. The bowels are as a rule confined. The illness lasts from a week to a fortnight. I believe the most likely cause is some form of ptomaine-poisoning, or some alteration or chemical change in the food, setting up a distinct form of poisoning. I may say in passing, in reference to food-poisoning, that I believe some of the rashes we meet with are due to this cause. I am not now referring to the common urticarial rashes from fish, tinned meats, etc., but to many indefinite rashes, which seem to recur over and over again, extending in duration for a fortnight or even more. It is interesting to note that our fathers and grandfathers constantly spoke of cases of gastric fever; and of late it has been rather the fashion to class the above with typhoid fever, and to say that the old diagnosis was wrong. It is well to remember that in days gone by, when laryngoscopes, ophthalmoscopes, stethoscopes, and all other scopes and speculums were unknown, there was much more time left for observation and thought, and old descriptions of diseases were very accurate in their general outlines. Now, the term "gastric fever" indicates the two chief features of the cases I have brought forward, viz. abdominal pain situated in the stomach region, and fever; hence I believe that many of these gastric fevers were truly gastric affections, and produced by some form of food-poisoning.

Cases of Difficulty of Breathing which require General Treatment.

Again, I would refer to some interesting cases in which we are primarily consulted for difficulty of breathing, but which do not improve or

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get well until a general course of treatment is adopted. The cases I am thinking of occur both in women and men, in ages between fifty and seventy. These patients are stout, indolent, well to do, and enjoy and partake of the good things of the table. On further inquiry you will find the shortness of breath is not permanent, and is often associated with a particular increase or unsuitability of food. On examination of the left heart it seems enlarged, impulse feeble, and the pulse is quick and weak; the bowels are constipated and albumen may be present in the urine. These patients sometimes complain of tenderness and weight over the liver and a general lassitude of mind and body; their nights are often sleepless or restless. On first sight one is tempted to give heart tonics and stimulants. I am not prepared to say that this is altogether wrong, but under these without other means the patient does not improve. The diet requires to be very carefully regulated, food given in small bulk, solid rather than fluid, and about every three hours. The bowels must be kept free; perhaps the most valuable drug at this stage is calomel, given in frequent and small doses. It is advisable for the first few days if possible to keep the patient in bed, as the free opening of the bowels may produce faintness and syncope if the patient is up and about. As soon as the patient is better moderate exercise should be insisted on, and a general plan of moderation in all things should be written down and pursued. The foregoing description will probably call up in your minds similar cases you have met with. I think that the breathing difficulty is brought about by the flabby condition of the heart setting up some dilatation, and this is followed by pulmonary and liver congestion, the original cause being the overloading of the abdominal organs from excessive or unwholesome foods, together with the absence of exercise or good air and the inability to dispose of the waste products. At any rate, my experience is that no amount of tonics, whether cardiac, nervous, or intestinal, have any real effect until the patient is put upon and acts upon a rational system of living. I should not, perhaps, have alluded to these cases except I had made mistakes myself, and seen them in the practice of others in which the value between the local and general troubles was not proportionately estimated.

On the Convalescent Stage of Disease.

In regard to the convalescent stage of disease, my opinion, from what I have seen in my own practice and in that of others, is that not infrequently patients are ordered to work before their health is fully established. The obvious answer to this statement is that in most cases time is too precious for the patient to spend very long in convalescence; but we must remember that convalescent homes are comparatively common, and admission to them by the poor is not difficult, and other classes often have opportunities, or could make them, for a longer rest after illness if the medical adviser urged it. It is only lately that I have fully realised how tedious is a full recovery after many cases of typhoid fever, and I will quote this disease as an example of many cases in which prolonged rest is required for recovery. What a serious exhaustion to the whole nervous system a case of typhoid must involve! A fever, almost continuously high, lasting often for four or five weeks, with a great portion of the bowel out of gear, all the internal organs more or less softened and worked at the greatest disadvantage. Is it a wonder that the convalescence must be and ought to be slow? The seriousness of the attack is seen later on in many ways, such as the loss of hair, the formation of nodes or abscesses, the feebleness of the muscular system, the slowness of the digestive organs to re-establish themselves, and the mental weakness or defective memory.

It is a hurrying age, and everything must be done quickly, and doctors, like other people, have fallen under this stress of example. Of course there are many exceptions to my statement; every case must be considered from what the disease is, and also the quality of it. I have made more mistakes from giving too short a time to a convalescence after acute disease than too long, and the excuse of the expense or value of time has not always been present. Where there is a morbidness of character or a likelihood of a long rest developing into a state of permanent idleness, then a different mode of treatment is advisable. Here, again, a broad view must be taken of the whole surrounding circumstances, and action carried out in accordance therewith.

July 9th, 1906.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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MORBUS CORDIS SINE BRUIT.*

By FRED. J. SMITH, M.D., F.R.C.P.,
Physician to the London Hospital.

GENTLEMEN,—First allow me to thank you for the pleasure of addressing you to-night. For it is a pleasure, though something of a task too; for I find that as a teacher of medicine one is inclined to be chary of putting one's ideas on paper, preferring the bed-side talks with students, and it does one good to be compelled now and again to deliver an address embodying a rather wider field of view than the single case, however interesting, affords.

In looking round for a subject your energetic Secretary, my old friend and fellow-student Dr. Hewland, offered me a rich choice of stirring titles, for he said, "We want stirring up a bit, Smith, we are in danger of stagnating in a medical slough of despond with surgical alligators always on the prowl trying to devour us and fixing us with a pitying eye as a poor set of helpless creatures that must at last fall a victim to the ever widespread maw of the operating table owing to the atrophy from disuse of those faculties of diagnosis and therapeutics which enable us to help our patients without the aid of the knife."

And, indeed, though his words may be intended as a little friendly badinage, there is underlying them a really serious idea well worthy of anxious thought and admirably handled in a recent article by Sir Dyce Duckworth. Look where we will at the three great cavities of the body, survey the world in general in all its classes, or consider the advance of modern science, and we find that the sphere of genuine medical art, that personal equation between doctor and patient, is everywhere being encroached upon—in one direction by the

* An address delivered before a combined meeting of the Brighton, Tunbridge Wells, Eastbourne, and Hastings Divisions of the British Medical Association, at Hastings.

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wholesale chemist and druggist, with his ever-increasing list of new chemical bodies guaranteed to exert with exactitude this or that effect upon our delicate physiological mechanism, totally irrespective of idiosyncrasy, constitution, or the vital capacity of the individual; on another side by the bacteriologist, the radiographer, and chemist, who with profound conviction in the mathematical certainty of their results reiterate with damnable persistency the cry that if you only find such and such microbe present, or such and such a radiographic picture, or if you only avoid such and such chemical body from your diet or your excreta you may be certain that the diagnosis is as follows, the treatment as plain as the nose on your face, and the result determined with the precision of a micrometer screw, and all this despite the facts that the patients still die, still feel gouty and unhappy, and do not go the way that a docile patient under such omnipotent laws ought to do. Then, finally, the medical horizon is clouded by the too easy way out of the difficulty by the surgeon, who, conscious, and rightly so, of the enormous advances his art and science has made, and aware of the great safety of modern operations, is a deliberate tempter to one's conscience when he whispers, "Let us look and see what is the matter; why bother yourself with weighing the pros and cons when a simple incision will solve all your perplexities?" And yet, gentlemen, even this omniscient wielder of the knife cannot always clear up a diagnosis for us, and we are still left, in some few cases, blindly groping for the cause of a severe abdominal pain. Of one case of this nature I personally have a very vivid recollection.

If these are not enough, we have also the blighting influence of a too narrow specialism. We have nowadays specialists for everything, not only skin, eyes, ears, nose, throats, uteruses, who are in some ways legitimate enough and have their distinct uses, but specialists for lung, heart, brain, and kidneys, who are, I think, distinctly less useful and very apt to miss the wood for the special tree that blocks their vision; and when it comes to specialists for gout, diabetes, and dyspepsia I must say that, in my opinion, the limit of reasonable specialism has been distinctly overstepped and we run a serious risk of leaving absolutely nothing to be attempted by the general practitioner or by the all-round consultant, unless, as I have heard suggested, these are to be considered the specialists for minor ailments, but no one knows what is a minor ailment.

Do not let me be misunderstood as railing at all specialisms; for where special modes of investigation are necessary, as in the throat, ear, nose and eye, and bladder, or where special minutiæ of operative ingenuity are required, only practice can make perfect; and it must at once be admitted that if advance is to be made in these directions, in technique, in dexterity, etc., the specialist is the only person who can make it.

Well, gentlemen, after this little polemic we must narrow our field a bit to the subject in hand, that of morbus cordis sine bruit—a subject upon which hitherto, I think I may say, not even a surgeon has dared to lay hands; for when it was proposed that a surgeon should dilate with a knife a stenosed mitral orifice, I think, though it was not specifically stated, it was intended that before the surgeon opened the heart he should have himself heard, or should have been credibly informed by someone who had heard, a presystolic bruit which was to be silenced by his operation.

In my case-books I find I have just a hundred cases labelled with this designation; they represent, naturally, a somewhat varied group in the particular set of symptoms for which my advice was sought, and also in the pathological condition of the heart which was present, the former ranging from sudden death through varying degrees of shortness of breath up to mere palpitation of the heart; and the latter from pure fatty degeneration up to mere neurasthenia of the very vaguest description, and it will be my endeavour to try to present you with some landmarks in diagnosis, prognosis, and treatment.

I would like, first of all, to offer a note or two on the condition in general. Thus there is a very sharp contrast between the cases we are discussing and those cases in which gross valvular disease is present or suspected because of (1) a bruit preceded by (2) a history of rheumatism or chorea, or accompanied by a history of hard work, or syphilis, or alcohol in persons of middle or advancing age; this group does not enter into my purview, except to offer an opinion that in the matter of giving an anæsthetic such cases are far safer than are those of which I am talking.

Then, again, there is another large group of cases,

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to which I believe the late Sir Andrew Clark was one of the first to draw attention, which in my case-book are labelled "bruits not morbus cordis"; *i. e.* they are cases in which a systematic examination and inquiry reveal a bruit with a history of some known cause for endocarditis, but the complaint they make is totally unconnected, so far as can be seen, with the mere derangement of circulation induced by a leaking or stenosed valve.

Diagnosis.—The actual diagnosis of the condition I am discussing depends on many factors, some of which lead us at once to the heart, which, indeed, is really only secondarily affected, such as emphysema and bronchitis with failing heart; these we may rule out of discussion, merely mentioning them as typical of the condition. Or, again, chronic renal trouble when the hypertrophy induced by that condition has reached its limit and the big heart has begun to find that its work is too much for its capacity, but a word or two on this may not be out of place, for it is at times difficult to discriminate between the effects of the renal trouble and those of the heart. The secret of discrimination lies in the examination of the urine for casts and the presence or absence of œdema. Again, take a man who has indulged so freely in alcohol as to have developed a cirrhotic liver and peripheral neuritis. I have seen several of such cases in which the complaints of the patient were mainly due to failure in the efficient driving power of the heart, due either to a disorder of the vagus nerves or to the direct effects of the alcohol on the muscle-substance of the heart.

Then for a third group in which the causation is obvious I may mention the specific fevers, more especially of course typhoid, of which we have all seen very many cases, and typhus, of which probably no one in the room has seen an example, though from the description of books it is obvious that heart failure is often the cause of death. Or perhaps a still more familiar example is the influenza heart. Who does not know the man who says, "I have always been short of breath since I had influenza, and occasionally I have palpitation of the heart very badly."

But these, after all, are hardly the cases I started out to identify. My intention was rather to describe those cases in which a patient comes complaining of a shortness of breath which he cannot understand or of an attack of pain in the chest which may have been anginal or may not, or again of palpitation for which there does not seem to be sufficient dependence upon the ingestion of food to offer as an explanation, or of an inexplicable asthma recently come to plague his life, of growing obesity, of insomnia, of conscious possession of a heart, of fainting attacks. How in these cases are we to recognise that the heart in its intrinsic mechanism of muscle and nerve is at fault? The answer is of enormous importance for the patient's friends and for the reputation of the doctor, for it is in these cases and not in valvular trouble that sudden death steps in to sneer triumphantly at the cheery prognosis of the thoughtless; or *per contra*, it is in cases mistaken for them that the threatened life outlasts the life of him who threatened, either being a *contretemps* which it is well we should avoid if care will do it.

The first thing to help us is the mere consciousness that morbus cordis sine bruit is a reality and a formidable one too. How often have I been told by the practitioner in practice, "I can't make the case out; there is 'so-and-so' which suggests cardiac derangement, and yet the heart is all right for I can hear no bruits." It is in this false security of no bruits that danger lurks. Perhaps he may be a more thoughtful man and adds, "Then, again, the apex beat is in its right place, so there cannot be any dilatation." But here comes in the second fallacy: what we feel and are accustomed to call the apex beat is produced, I am quite sure, by many different parts of the heart other than the apex of the left ventricle. In some cases it is produced by the middle or base even of the walls of that cavity, often again by parts of the right ventricle, and I do not feel sure that in certain cases it may not even be an auricle producing an outward and visible impulse.

Next place your whole hand on the chest, not the tips of a finger or two, but the whole palm, including the thenar eminence, and put your brains and minds into your hand. You will frequently appreciate a good deal more than a mere apex beat; it is simple to appreciate a big heart working away under a covering of emphysematous lung (the opposite condition of a retracted lung is not worth more than a passing mention, for its history is, as a rule, too obvious).

Percussion of the relative or deep dulness of the heart should be practised as a routine

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measure to corroborate or negative what can be suspected from palpation, though I must confess that it is but rarely I resort to this method of investigation, believing that it is rather trying to the patient and open to a good deal of criticism from the point of view of the personal equation as to the delicacy of perception of the observer's ear.

We then betake ourselves to the aid that the stethoscope may yield, and it is here that nothing but care combined with experience can help, for we have *ex hypothesi* excluded a bruit, and this is the only thing that most students care about.

A student has examinations looming ever larger and larger on his horizon, and though one may in the wards endeavour to teach him something more than bruits it is quite impossible in the nature of the cases to take to the examination-hall anything more than a bruit.

But it is the other points that we must now take account of. I might here say that my invariable practice in home work is to listen to every heart as a routine practice for a full twenty seconds, and in every case of suspicion for much longer, and not only so, but I listen for a full twenty seconds in the standing, sitting, and lying positions, and before and after walking or trotting (if possible) several times up and down my consulting-room. By taking this amount of care and time one can generally get so far as to say "This heart quickens too much under exertion, and does not slow down properly with rest," or "This slight irregularity becomes more accentuated than it ought to do under strain."

The converse case must also not be lost sight of, mostly occurring in young subjects, say from sixteen to twenty-two, who say they get palpitation and are "frightened to death" about their hearts, the cardio-neurasthenic, or cardio-neurotic. This class of patient must be discussed a little more fully under "Treatment."

Excluding, then, *ex hypothesi* anything in the shape of a bruit, what are we to look out for, or rather, listen for? Well, the first thing is the actual frequency of the beat and its variations, as detailed above, under conditions of rest and exercise; then the rhythm—a difficult thing to define, for it includes, not only actual gross irregularity or the occasional, perhaps regular, dropping of a beat, but some intangible character in the sounds themselves and a most exact appreciation of the pauses between the beats.

Then the contrast between the two sounds themselves; the old "lub dup" of our student days is the best guide, and the more this tone distinction between the two sounds is diminished the greater must be our suspicion.

The actual bulk of noise—if I may use such an expression—is also a valuable guide; by this I do not altogether mean simple audibility, for a whispered word, to suggest an analogy, is often quite as audible as a spoken one, and it is easy to distinguish the long "lub" of a healthy big heart acting behind a screen of lung from the first sound produced by a weak heart which is being over-strained, provided one has made a practice of thinking of such things.

These are the chief points to be ascertained by auscultation—frequency, character of rhythm, irregularity or regularity, the loudness of the sounds, and, above all, the due preservation of the contrast between the two sounds. In the febrile heart, for example, while both sounds may be weak, the contrasting difference in length is almost always appreciable.

Prognosis.—This is always grave, and must necessarily be so, for we are considering one of the gravest affections that can occur—viz. an intrinsic degeneration of the nervo-muscular structure of one of the three great pillars of life. But it must, all the same, not be considered to be hopeless; take diphtheria, typhoid, influenza, for examples. We know here that, at any rate in the young and middle-aged, we are dealing with a condition from which recovery of the heart is the rule and death only the exception—a fact which proves to a demonstration that the nerves and muscle of the heart are capable of recovering their pristine energy; and when we meet with a similar condition in advanced age, or without such an obvious cause, we must not assume that the capacity for a *restitutio ad integrum* is totally wanting. But all the same, in a man over, say, forty or forty-five, the prognosis is an extremely anxious one, and the patient must be guardedly warned of the possible effects of not following advice, and some one of his friends must be spoken to more openly and told straight out upon what a slender thread life hangs, so that they may be quietly endeavouring to persuade the patient to make a will, etc., etc.

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Treatment.—Of those cases where the heart is obviously only secondarily affected I do not propose, nor am I able, to give you anything very novel or startling; the only hint I can offer you there is to ask yourselves whether you bleed your patients as frequently as you think you ought. I am well aware of the difficulties connected with this operation, for even as a consultant I feel that I have scarcely done my duty in this respect, having frequently given in to the specious suggestion of the practitioner that if the patient dies the friends will say it was the bleeding that weakened him so, and if he gets well they will say it was unnecessary.

But in all these cases we are dealing, so far as we can tell, with hearts which have intrinsically sound muscle structure, and we can hope, when the immediate excess of strain has been removed, the organ as a pump will recover.

The cases I am more immediately dealing with are those in which we believe either the nerve or muscle, or both, to be essentially weak in themselves; how dangerous any little extra strain can be my records show very distinctly, for against at least ten of my hundred cases is the ominous record "found dead," "dropped dead suddenly," etc., and hence it is quite obvious that our first point is to *order* the patient not to go in for tram or train catching, never to hurry suddenly for any purpose, and not to strain. It is here that laxatives have their value in preventing constipation with its strain. This advice may be followed for the rest of the patient's life, but in the acuter cases—*e. g.* after typhoid, diphtheria, etc.—you may well say "But the patient cannot go on in bed or avoiding strain. How shall we know when to let him up or relax his care?" I have my own little habit for that, which is to listen to and count the heart beats, lying, sitting, and standing by the bed, remembering that the sitting should quicken it by about ten per minute and the standing by another ten. I then allow a rough average of a quickening of twenty for each, and if I get anything much over this I say we will have another day or two's bed and idleness, and when finally we think that the bed may be exchanged for the couch we must make the change very gradually, beginning with, say, an hour, and prolonging the period according to the feeling of the patient.

On theoretical grounds digitalis should be contra-indicated in those cases where we believe that fatty, or fibrous, or other form of actual degeneration of the muscle is present, induced as it is by atheroma of the coronaries (it is extraordinary what a very frequent post-mortem finding is atheroma of the coronaries), but as a matter of experience I find that I get a good deal of benefit in such cases from its use in small doses, say 3 or 4 minims as a maximum of the tincture or (what I usually find much more satisfactory) ℥j to ℥iss of the infusion, or perhaps even better still, gr. 1 of the powdered leaves as in the old Bailey's or Guy's pill.

Of all drugs, however, for the condition by far the most useful are the iodides. I tried for a little while the sodium salt, but I cannot say that it has any superiority over the potassium one. I do not believe in potassium having any specially depressing action when used in ordinary medicinal doses, but I must, of course, admit that there are certain people who have a special idiosyncrasy with regard to potassium iodide, and assert that they cannot take it in any dose at all. I am open to be convinced, but I want convincing, that the man exists who cannot take it in half-grain doses, and surely if they are so susceptible it is probable that this small dose would give as good effects.

I said just now that I would refer again to the question of age under the heading of "Treatment," and really it is here that age has its most appropriate spot for mention, for the treatment depends on diagnosis, and diagnosis has perhaps for its greatest factor age, beginning with the small child with congenital morbus cordis sine bruit for whom no treatment is of avail, unless forsooth the enterprising surgeon will suggest a plastic operation for closing one opening and enlarging another. In young children and adults up to, say, thirty-five or forty, the condition I am discussing very rarely occurs without some fairly obvious cause, and it is now almost a truism to say that the best treatment of any disease is to remove the cause. The causes which we may have at work are, for instance, self-abuse, which often brings a young person to a medical man with symptoms strongly suggestive of heart trouble, and even in reality dependent upon an upset of the nervous mechanism of the pump. Or, again, over-smoking, of which I remember one very typical example in a young Frenchman who wished to insure his life, but with an ordinary pulse-rate of 150 I could not pass him; omission of

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tobacco soon restored the rate to limits which were more reasonable, though even then his heart would quicken unduly on exertion. Alcohol in excess may produce heart trouble, possibly of the same nature as the tremors so frequently present in the toper's hands. Syphilis, again, either as a specific fever or as a tertiary affection, may be at the bottom of our diagnostic puzzle and require treatment by mercury. On this perhaps you will pardon a slight digression. One of my surgical colleagues said to me not long ago, "Smith, what cases of tertiary syphilitic affection do you treat with mercury?" My reply was prompt, inclusive, and decisive: "All, without exception, as I consider that a tertiary affection is a proof that the previous treatment only scotched and did not kill the *Spirochæte pallida*." In these younger subjects take, again, digestive troubles; these are common enough as causes of what to the patient is a serious heart trouble, and to read the recent article on "Alimentary Reflex," in the 'Lancet,' it would seem that in the elderly they may be real causes of what to medical men is dangerous heart mischief.

July 16th, 1906.

Rise of Temperature from Suggestion in the Tuberculous.—Lorenz relates his experiences with a number of tuberculous patients who were given sham injections of tuberculin. His tables show an almost regular increase in temperature after a sham injection in 44 out of 200 persons tested. The excitement and the suggestion send the temperature up, although none of the supposed tuberculin was really injected. His experience suggests that it might be wise to test the influence of suggestion in this way before really injecting the tuberculin later. A rise of half a centigrade degree can be accepted as a positive reaction to the tuberculin. It should be computed from the temperature noted after the sham injection, allowing for the rise due to the effects of suggestion. The sham injections were liable, not only to send up the temperature, but also to affect the general health. It was noticed sometimes that those patients who had the most pronounced rise of temperature after a series of sham injections failed to show any rise when a genuine injection was given. Another fact noticed was that the suggestive reaction was generally more pronounced when the organism was depressed from any cause, a slight cold or inflammatory process in a tooth or the like.—*Journ. A. M. A.*, vol. xlvi, No. 25.

TWO CLINICAL LECTURES

ON

TUBERCULOUS ARTHRITIS.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

Lecture II.

Gentlemen,—On the last occasion we were discussing tuberculous arthritis and the methods by which we might ascertain that the swelling was still within the capsule of the joint. Perhaps we had gone beyond that, because I remember mentioning the nature of the swelling inside the capsule of the joint, and I briefly referred to what might ensue if the swelling escaped from beyond the capsule of the joint and passed into the tissues of the limb and was allowed to escape amid sepsis, or was opened without perfect aseptic precautions. The swelling within the capsule of the joint is easiest to see at the periphery of the limbs. For instance, it is quite easy in the ankle and the wrist, not difficult in the knee and in the elbow, but exceedingly difficult in the shoulder and the hip, and also in the joints of the spine and in the sacro-iliac articulation. The reason why it is easy to see in the peripheral joints and difficult in the proximal joints is simple; it is simply this, that in the case of joints like the wrist and ankle their capsules are merely covered with tendons and thin tissues. The elbow and the knee are rather more difficult, because, especially in the case of the elbow, muscular bundles surround the joint. The shoulder, of course, is very difficult, because it is a small joint, hardly bigger than the elbow and the wrist, and is overlapped, as you are aware, with exceedingly strong muscles. Much more is this the case with the hip, which is likewise a small joint; it lies at a great depth and is surrounded by the largest muscles in the human body. But even in such a difficult joint as the hip, if your eyes have been trained to recognise the hills and hollows in its vicinity, you will have no difficulty in observing the slight obliteration of the fold of the groin. Or you may be able to see that the common femoral artery is lifted up. The hip-joint is, of course, peculiar in this respect, that its capsule is thin

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behind, but where it might easily be supposed to be bulged by swelling within the joint the capsule lies at a great depth and is hidden by the gluteus maximus. The front of the hip-joint is not easily bulged, because it has in it the strongest ligamentous band in the whole body, which you may therefore infer cannot be easily lifted up by a swelling or effusion within the joint. Nevertheless, in spite of that, a little care and attention in examination will always show you that the common femoral artery is lifted up by the front part of the capsule of the hip-joint. However, the great joints of the shoulder and hip, with their great muscles, display more characteristically than other joints one of the essential diagnostic signs of arthritis, and that is the wasting of the muscles. In the case of the hip the wasting of the gluteus maximus is quite obvious. But do not suppose that the wasting over the nates in hip-joint disease and the muscle above and below the joint is the only thing which you have to go upon. In addition to the wasting you have the flexion, and in addition to that the limitation of movement, ultimately ending in rigidity or in fixation. When it is a question as to the limitation of movement in any joint, proceed with extreme caution. I think that a person who unnecessarily hurts a human being is either heartless or foolish. I would suggest that when the question of the mobility of the hip-joint is to be ascertained the finger should be placed on the anterior superior spine of the ilium and the patient be requested to flex or extend the joint. You will see at once whether the pelvis moves with the thigh-bone. When you yourself undertake to ascertain the limitations of the movements of a joint, you should proceed with extreme caution. Begin on the healthy side and observe its movements. You will begin, of course, with flexion and extension, next you will try adduction and abduction, and lastly you will take the rotation and examine that. Why this order? For this simplest possible reason. When a joint becomes inflamed that is the order in which its movements are lost. The first movement to be lost is rotation, the next movements to be lost are adduction and abduction, and the last movements to be lost are flexion and extension. I have known this occur: A man is thrown from his bicycle; he falls and injures his shoulder-joint, and he has a violent attack of synovitis in that joint. At last that is thought to have subsided, but it is found that the shoulder-joint is exceedingly stiff. His medical man undertakes to move it, but he causes so much pain by the attempt that the patient very naturally leaves him and goes to a medical man who is better acquainted with his business. It is ascertained that this man has perhaps half of his proper amount of flexion and extension of his shoulder-joint forward and backward. He has not abducted the shoulder-joint because of the pain, and rotation was never attempted. An exceedingly shrewd man was this patient, who had made his fortune at growing hops, so you can imagine how exceedingly capable such a speculator must have been. So he at once observed, "My doctor began by turning it round, and he of course hurt me dreadfully." Adieu to that patient! There is another sign besides limitation of movement, fixation, and rigidity to assist in the diagnosis of arthritis, and that is the tenderness which can be elicited by pressure. This pressure, again, requires to be applied with extreme caution. I often see it applied in a promiscuous manner. If a patient has an inflamed synovial membrane, in a joint such as the wrist-joint, then that joint ought to be painful and tender in all its parts. For instance, if the tenderness on pressure were confined to the back of the wrist-joint you would be suspicious about it. But if the tenderness on pressure was elicited, not only on the dorsum of the wrist-joint, but also by pressure upon the flexor surface of the joint, also by pressure on the radial side of the wrist-joint, also by pressure on the ulnar side of that joint, there would be, surely, growing up in your mind a strong conviction that there was something tender inside the whole of the capsule of that joint. In some of the joints, as I remarked, the signs of swelling are exceedingly difficult to elicit, and that is especially the case with the vertebral column, which, after all, is nothing more or less than a long complicated joint. When you examine the vertebral column which is supposed to be the seat of tuberculous disease and of tuberculous arthritis, you will observe that of which I have already been speaking, first of all, the very marked wasting of muscles on either side of the spinous processes, and lastly, limitation of movements in the inflamed part of the vertebral column. In this disease in the earlier stages limitation of movement is not

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particularly easy to discover. Perhaps it is that one does not always know how to do it. I am accustomed to do this, to go methodically over the different vertebræ. I put my four fingers on four spinous processes, and then request the patient to flex and extend the vertebral column. You will observe that in the parts of the vertebral column which are not diseased the fingers come apart—that is to say, the vertebræ go apart during flexion and come together during extension. In the part of the spinal column which is inflamed, or which is the subject of tuberculous disease, there is absolute rigidity, exactly the same as that which you find in any other tuberculous joint in any other part of the body. There are a few other curious symptoms which are associated with tuberculous arthritis, and I ought not to pass over the curious starting pains which occur when the patient is going to sleep. You must not think these are peculiar to tuberculous arthritis. They are seen in connection with any acute inflammation of a joint. I have often observed them, for instance, after wiring of the patella, during the time that the joint is inflamed, as a result of the effusion of blood into it caused by the operation, perhaps, or perhaps due to the injury to the synovial membrane caused by the sponging and manipulations and lotions. Also these pains are exceedingly acute in septic arthritis. These starting pains are supposed to be indicative of changes taking place in the articular cartilages—that is to say, commencing destruction of them. I believe that that is probably a truthful statement. But I would say this, that having watched this question to the best of my ability, I have not infrequently observed when I came to operate upon a patient who had had very severe starting pains in the joint that there was exceedingly little destruction of the articular cartilage. I have sometimes observed, too, when the starting pains have been slight or absent that there has been a great deal of destruction of articular cartilage. So that to my mind there is something behind this question, with which I personally am not acquainted. As regards this destruction of cartilage in tuberculous arthritis, I would just make one remark to you. There are many methods by which cartilage becomes removed in the human body. Here I show you a patella from a case of osteo-arthritis, in which you observe there is removal of cartilage. Here, again, is a tuberculous knee. In each of these the cartilage is beginning to depart. But observe how it is going. In the case of osteo-arthritis in the patella it is beginning to go in the centre, in the case of tuberculosis of the joint it is beginning to go at the edge. I wonder if I am right in my way of thinking of that. It is this: that osteo-arthritis is to a large extent, and in the main, a degenerative disease, and the disappearance of cartilage is a process of degeneration due to insufficient nutriment, and therefore takes place at the centre of the cartilage, which is farthest from its nutritive supply. But the destruction of cartilage which takes place in tuberculous and suppurative arthritis is an active process, and therefore begins where the vascular supply and changes are most active. Around the articular cartilage of all joints there is a corona of blood-vessels, the corona vascularis I remember it to have been called, and there the conditions are best suited for an active inflammatory process to take place. This removal of cartilage I will refer to again in a moment, because it has an important bearing on the matter of surgical treatment. The general symptoms of a patient with tuberculous arthritis ought to be studied by you, because of course such symptoms have to be watched. They may indicate that the patient is progressing in an unfavourable direction, or their subsidence may indicate that the patient is progressing in a favourable way.

First of all with regard to the pulse in people who have not only tuberculous arthritis but tubercle generally. I wonder if it is correct to believe, as I believe, that the tuberculous subjects have rapid pulses. At all events, I observe that in our wards the children with tuberculous arthritis have rapid pulses, and continue to have rapid pulses quite apart from the presence of suppuration or of sepsis, or even of progressive changes. The temperature, obviously, in cases of tuberculosis is usually elevated, and in the general principles of treatment of disease you have to think twice before you allow a patient to be moved out of bed and put into the open air when the temperature is elevated. We have not studied very much the condition of the blood in those who have tuberculous arthritis, but it is customary now, when the question of inflammatory and suppurative changes arises, to make a blood-count. Our experience is that there is always a certain leucocytosis. Of late the journals have

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been full of the method of examining and estimating the condition of the blood by ascertaining the opsonic index. I do not know enough to be able to inform you upon this matter; you ought at all events to endeavour to learn about it. We have had the opsonic index of tuberculous people estimated for us in the ward, and we have had them treated with appropriate serums, but I myself have not yet observed that any good has accrued from this method.

Now we will consider for a little while the progress of a case of tuberculous arthritis under the most favourable conditions. Supposing this patient were not treated at all but left, as the children of the poor not infrequently are, to their fate. The almost inevitable sequence of events would be this: that the joint would become flexed, it would be perhaps displaced and possibly even dislocated. The rotation outwards and displacement backwards of the knee are perfectly obvious. The hip not infrequently becomes flexed, abducted, or adducted, and perhaps the head of the femur may become displaced on to the dorsum of the ilium; in fact, there may be a true dislocation. Of course the wasting of the muscles persists, so that the leg is exceedingly thin. If dislocation does not take place, and if the disease proceeds, the probabilities are that the joint, quite apart from suppuration, will become firmly ankylosed in a flexed position, and in the case of some joints may be rotated or abducted or adducted, at all events altered. When suppuration occurs more may happen. If the patient were to survive the sepsis which may spread from the abscess outside the joint to the interior of the joint, and if there were no necrosis or progressive caries to carry the patient off, then something else might happen. The process of suppuration is associated with the formation of granulation-tissue, which in its time removes the whole of the articular cartilage. When the whole of the articular cartilage between, say, the femur and the tibia and upon the surface of the patella had been taken away, then the opposing granulation-surfaces might unite. Then there might be an organisation into fibrous tissue, or possibly the granulation-tissue might ossify. If that were to take place with the joint flexed, or rotated, or abducted, obviously the patient would not arise from that very long illness with a particularly serviceable limb. I suspect that it would be a limb which would be absolutely and entirely useless to the patient until it had been straightened by a surgical operation, or, indeed, even to enable him to get about the world in a mutilated condition, it might have to be removed. Presently I shall refer to this again, because I am really conveying to you some of the principles upon which the excision of joints is founded. Observe how you are to proceed in a case of tuberculous arthritis. If the flexion is not already there, though it probably will be when the patient comes to you, obviously you have to prevent it from occurring. Observe how that is usually done here. If the joint is flexed gradual extension is applied to it, and by degrees it is brought straight. I am assuming we are speaking of the lower limb. Not only is that done, but at the same time elaborate attention has to be paid to the general hygienic condition of the patient with regard to light, fresh air, cleanliness, warmth, good food, and so on. Observe that if I am right in saying that light is so important to the patients who have tuberculosis, obviously the gloom of great cities is not altogether the best for them. Next, if it is so essential that they should have fresh air and proper ventilation, the ancient hospitals of ancient cities are not very suitable places. The other necessaries, of course, can be provided anywhere. But perhaps the case has proceeded farther than we are supposing. The joint is flexed, and it is perhaps displaced. Possibly it has undergone some fibrous or bony ankylosis, and then arise the various questions of surgical treatment. The ordinary treatment which would be proposed for a joint in that condition which would not yield to gradual extension would be an amputation or an excision. I thought I would refer to this because I am more usefully employed in trying to convey to your minds the principles of surgery than in telling you of the details of individual cases. Now, if a surgeon undertakes to resect a tuberculous joint, he must, as a preliminary, make up his mind to clear away the whole of the disease. Then, after the removal of the whole of the disease, he has to make up his mind that the patient has to be left with a useful limb. Now, a useful limb is not the same thing in the arm as it is in the leg. I think a person might have a useful fore-limb if the wrist-joint were ankylosed. The wrist-joint movements are not very extensive, I doubt if a person would have a very useful arm

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if the elbow-joint were ankylosed; in fact, I am sure he would not, for the reason that patients with ankylosis of elbow-joint have excision performed to give them a movable elbow-joint. I am also sure that an ankylosed shoulder-joint is not very useful to its possessor, because I have known patients have their shoulder-joint excised so as to give them a movable shoulder-joint. But with regard to the knee, I think you may be clear in your minds that the excision would be almost a total failure unless it resulted in firm bony union between the tibia and the femur. The hip-joint is on rather a different plane in this respect. Excision of the hip-joint is not, to my mind, a very successful proceeding in surgery. An ankylosed hip, if you can get it ankylosed by bone, leaves the patient with a most dreadful gait, and if the hip-joint is not ankylosed, and the head of the femur in the process of excision, of course, has to be removed, think of the patient's condition then. He has now the remains of the neck of the femur above the acetabulum on the dorsum of the ilium, and he is very much in the position of a patient who has a congenital dislocation or displacement of the head of the thigh-bone. That is a condition which all sorts of operations have been devised for, and methods of treatment have been tried which keep the patients recumbent, with their legs spread out in an extraordinary manner for two or three years. Excision of the hip surely is not a triumph of surgery. Observe what you wish to achieve—first of all the removal of the whole of the disease, next either a movable or a fixed joint.

First, as to the removal of the whole disease. Now, you must understand that the removal of the whole of the synovial membrane of a joint is a very tedious proceeding. To remove the synovial membrane of the knee-joint it takes, I should say, at least three quarters of an hour to an hour. The process is speedily carried out on the front of the joint, where the synovial membrane is easily reached, but it is by no means easily carried out behind the condyles. As the whole of the synovial membrane must be assumed to be diseased, it has to be followed in its various ramifications. Sometimes the prolongation along the tendon along the popliteus is most difficult to remove, and the parts which go behind the condyles and underneath the gastrocnemii. I do not think the prolongation beneath the crureus is very difficult to remove. But remember that every piece has to be tracked out and taken away. I want to strongly emphasise this necessity because I observe that gentlemen when they are reading their anatomy learn the prolongations of the knee-joint very much by rote for examination purposes. They do not know that the day might come when it might be a matter of extreme importance to them to know how to be able to trace them accurately and properly. I have known cases in which the synovial membrane has been left behind the condyles, and in consequence the disease has had an opportunity of continuing and relapsing. I want to draw your attention to another thing about synovial membrane. If you leave portions of it behind in a joint which you think you have excised, you will find that the case may not do particularly well. First of all, that synovial membrane is very likely to be tuberculous, and the tuberculous process will go on in it. In the next place synovial membrane is a curious kind of tissue. It is always classed in my mind with mucous membrane. Have you thought how difficult it is to deprive any channel in the body of its mucous membrane or mucous lining? But as long as a scrap of it is there it will continue to secrete and keep that sinus open. Again, synovial membrane when it is acutely inflamed will pour out enormous quantities of fluid, and it will be a long time before that discharge ceases and the diseased membrane becomes replaced by a fibrous mass. In these tuberculous joints if you leave synovial membrane behind not only may you be sure that the disease will continue to progress there, but you may be sure that the membrane will remain there for a long time and will continue to pour out fluids which will jeopardise the result of your operation. In our efforts to remove all the disease we come to the next stage. There may be disease, not only in the membrane, but in the bone. Because in at least half the cases of tuberculous arthritis there is tubercle in the bone which during the operation can be seen with the naked eye just as well as tubercle can be seen in the cancellous tissue of this spine. It may be necessary to remove tuberculous bone, but that would be an act of judgment at the time. But it is not likely that you will be able to remove tuberculous bone until you remove the whole of the articular cartilage. Articular cartilage is a curious structure which has been put between the ends of those

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bones to prevent them sticking together, and so that they may slide easily upon one another. So as long as articular cartilage exists between the ends of the bone they will never join together by bone. So that clearly you have to remove the whole of the articular cartilage. Perhaps it may be done by saw-cuts, or by gouges, or by forceps, but all of it ought to come away, every scrap of it. These two points are essential, whatever the ultimate result has to be. The whole of the disease has to be removed, and obviously the whole of the cartilage. Your object is either to secure bony union or to secure mobility. A rule in surgery is your guide. If it is your intention to have bony union, then you must remove as little bone as you possibly can, and you must keep the bones in most exact apposition. Here are two radiographs which were sent to me last week. You can see how those principles have been carried out in that knee, which was resected. The excision which I do is this: I cut the femur as in the sketch, and

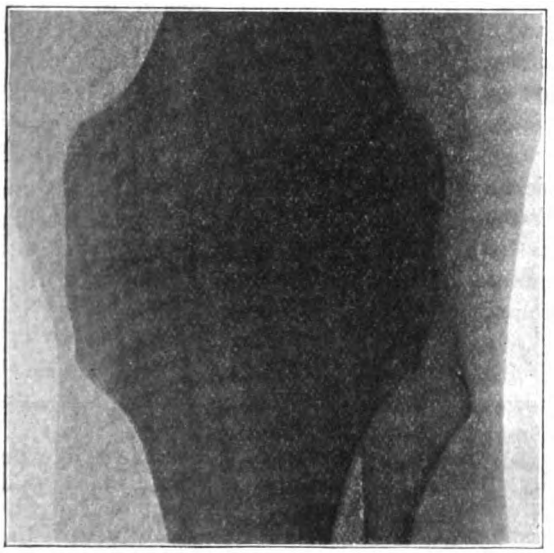

FIG. 1.—The femur is above the tibia and fibula below. The shadow of the patella can be made out. The oblique cut is fairly indicated.

then the tibia in the same way, and they wedge into one another. They cannot slide sideways and they cannot rotate. They might slip backwards, but the back of the femur and the back of the tibia are properly applied to a splint, from which they do not move. To make the security a little greater, observe that the patella is kept. The articular surface of the patella was removed, and it was put down upon the cut surface of the femur and tibia and fastened there by an ivory peg. So there is as little bone as possible removed. I do not see how you could remove less bone and get so much bone in contact. The bones are kept perfectly

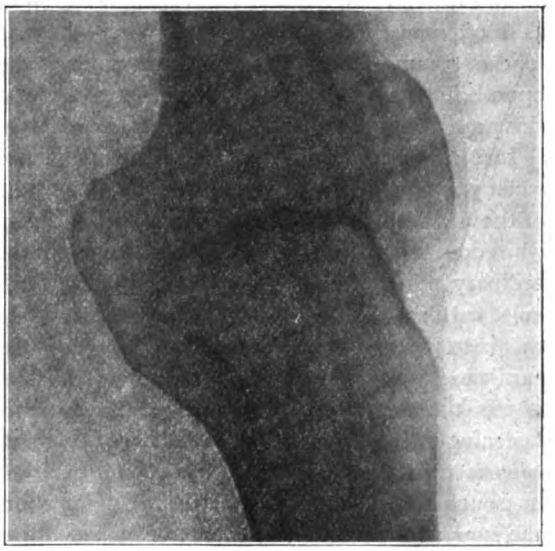

FIG. 2.—Femur above, tibia below, patella to right hand. The shadow of the ivory peg can be seen through the centre of the patella and entering the tibia.

quiet upon one another until they have stuck. If you look at these skiagrams I think you will agree that there must be exceedingly strong bony union between the femur and tibia and between the patella and femur and tibia.

Now observe what next is done to get bony union. At the operation you remove the whole of the disease, the whole of the articular cartilage, and as little bone as possible. You then arrange to keep the limb as still as possible. This excision of the knee was kept absolutely still after the operation for at least six weeks, and then the patient had a suitable apparatus before he was allowed to go about in any way whatever. He was not allowed to place any weight upon that limb until it was certain that he had bony union and that it could not be bent. How do you ascertain when an operation such as this has resulted in bony union? It is quite simple. When bony union takes place there is not only immobility, but there is also an entire absence of pain at the seat of the excision. A patient has broken his thigh-bone,

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how shall you know whether it has united? You try to move it, but you cannot. But he says it hurts very much. That means it is not united by bone, but by fibrous tissue. Absence of pain on movement shows that it is united by bone. Fibrous union is always painful, it is always troublesome for the patient, and it is always laying him up. And, as I often heard Sir William Savory say—he was fond of sonorous language—"Fibrous union is the opprobrium of surgery." Finally, you have a skiagram traced as in this instance.

I will now refer to the surgery which you practise when you want to get a movable joint. The whole of the disease has to be removed, and the person who does not do that does not understand the ordinary principles of surgery. Next, all the articular cartilage, I think, ought to be removed. In this case you wish to get movement. But articular cartilage is not very satisfactory stuff. As long as it was there it might have prevented the bones fastening together, but articular cartilage is a tissue of very low vitality, and it might become necrosed or troublesome. It may be that I am talking about the surgery of pathology of twenty years ago; I may be wrong, but I have been brought up with a prejudice against the presence of articular cartilage in wounds. That being granted, how will you proceed to fulfil the next condition which we laid down, that you wish to have a freely movable joint when your excision is over? You do not do in the wrist, the elbow, or the shoulder what you do in the case of the knee. You do the exact reverse. You remove a great deal of bone, and begin to move the bones upon one another as soon as you possibly can. I have not been very successful in my excisions of the shoulder, the elbow, or the wrist. I believe the reason I have not always been very successful with them is this, that I have been rather afraid to remove a sufficient amount of bone; I have not hesitated to remove one and a half inch of the humerus, and have not hesitated to remove over one inch of the ulna, but I confess I have not felt satisfied with the result. One reason, perhaps, is this, that the patients who come to the hospital and who have a joint excised have not enough intelligence and not enough fortitude to understand that that joint, if it is to be movable, must be moved freely and much; and, therefore, as soon as the attempt is made to move the joints, which are rather painful, they discover that they have relatives who are dying, and so must leave the hospital. They ultimately come back about two months afterwards with a joint which is not at all good, which may move, but does not move in a satisfactory manner.

I have been telling you something about the treatment of tuberculous arthritis, the palliative treatment and the active surgical treatment. But I think it must occur to your minds that I have not tried to put a gloss on the matter at all, I have told you that my own particular attempts have not been very successful, and, in fact, I do not think that the surgical treatment of tubercle can be exactly reckoned as a surgical triumph. However, there is this to be said about tubercle, that it is a preventable disease, and it is probably to be prevented by the proper supply of light, of ventilation, of cleanliness, and warmth and food. And, moreover, it is probably to be prevented by the segregation of tuberculous people, and lastly, by the immediate slaying of all tuberculous domestic animals. Besides that, it is obvious that tubercle and probably other diseases, such as carcinoma, must be prevalent where people are overcrowded, and especially must tubercle be common in communities where the inhabitants marry exceedingly early and breed a number of sickly and ill-developed degenerates. I am mentioning this purposely, because the future is going to be in your hands, and you have to think all these things out and see how the health of the community can be improved.

July 16th, 1906.

Spread of Tuberculosis by Way of the Lymph.—Haentjens has long proclaimed that tuberculosis is primarily an affection of the connective tissue and that it spreads by way of the lymphatic system. He regards this conception as very important, for if the danger for the organism from tuberculosis lies in the connective tissue and its juices, it is here that the battle against the tuberculosis virus must be waged. Proceeding on this assumption, he has been producing a prepared curative serum by inoculating animals with specific tuberculosis virus obtained from the blood-forming tissues and bone-marrow, thymus juice, spleen extract, and lymph. His serum displays both preventive and curative properties. He is preparing a detailed report for publication later.—*Journ. A. M. A.*, vol. xlvi, No. 25.

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A CLINICAL LECTURE

ON

PROLAPSE OF THE UTERUS IN PREGNANCY.

Delivered at the London Hospital.

By HENRY RUSSELL ANDREWS, M.D., B.S.Lond., M.R.C.P.
Assistant Obstetric Physician to the London Hospital.

GENTLEMEN,—I have chosen the subject of prolapse of the uterus during pregnancy because we have had several cases of this condition in the gynæcological out-patient department recently and I have found that students are always interested in the treatment and prognosis of these cases, and do not seem to be able to find very much about them in text-books.

Cases which are diagnosed roughly as "prolapse during pregnancy" must be divided into two classes:

(1) Cases of prolapse of the uterus.

(2) Cases of hypertrophy of the cervix.

(1) *Prolapse.*—Women with severe degrees of prolapse, viz. those in whom the whole, or a large part, of the uterus is outside the vulva, are less likely to conceive than those in whom the cervix is inside the vagina, but pregnancy occasionally occurs in the former class, as in the case of A. C—, whose notes I will read you later.

In most cases of pregnancy with prolapse the symptoms due to the prolapse are increased during the first two or three months of pregnancy; *e. g.* there is great frequency of micturition, there may be a considerable amount of discomfort from sense of weight, and if the cervix is outside the vulva it is likely to become swollen and ulcerated, the œdematous swelling being very great sometimes. As a rule, by the end of the third month or a little later, the body of the uterus rises out of the pelvis and the cervix is drawn up into the vagina. If, however, a large part of the uterus is outside the vulva the body may remain in the pelvis, and as it is getting bigger than a fœtal head symptoms of incarceration occur, ending in abortion if the condition is not relieved.

(2) *Hypertrophy of the cervix.*—As you all know, in prolapse of the second degree—*i. e.* when the uterus is partly inside and partly outside the vulva—there is usually elongation of the supra-vaginal portion of the cervix to such an extent that the sound passes four and a half to five inches.

Hypertrophic elongation of the cervix, however, the second condition which we must consider, affects the vaginal portion of the cervix and may resemble or be accompanied by prolapse. In these cases pregnancy causes increased swelling of the enlarged cervix, and if the vaginal orifice is small the part of the cervix outside the vulva may be so much swollen that it cannot be pulled up. In these cases unless the supra-vaginal portion of the cervix becomes greatly stretched and elongated the body of the uterus will be unable to rise up out of the pelvis.

The chief difference between the two conditions lies in the fact that prolapse is rare in multiparæ, while hypertrophic elongation of the vaginal portion of the cervix may occur in such patients and give rise to trouble during the first pregnancy.

Clinically the two conditions may be considered together. In one case, E. E—, the notes of which I will read later, the condition might well have been considered to be due to hypertrophic elongation of the vaginal portion if we had not known that the descent of the cervix had occurred for the first time after her first confinement, and that the protruded cervix had not begun to swell until after the beginning of her second pregnancy.

Some of you saw a patient in the out-patient room a few days ago who said that when lifting a heavy weight "something gave way inside and the womb came down." Such cases of acute onset of prolapse are not very common, but a similar thing may occur during the first two or three months of pregnancy, prolapse of the pregnant uterus being produced by a violent strain or a fall.

These cases may be considered with those in which pregnancy occurs in a prolapsed uterus.

SYMPTOMS.

As I said before, the symptoms due to the prolapse are usually intensified during the early months of pregnancy, but after the fundus has risen up above the pelvic brim the symptoms in most cases disappear, and the patient does not suffer from prolapse until after labour. There may be some prolapse of the vaginal walls in the later months, but this does not usually cause much trouble. A

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patient came to the hospital about two and a half years ago complaining of "great soreness of the private parts." She was about seven months pregnant; the cervix was in the normal position, but the anterior vaginal wall protruded from the vulval orifice and was much congested and slightly ulcerated from rubbing against the clothes. A few days' rest in bed and application of lead lotion relieved her discomfort, and she subsequently wore a napkin to prevent recurrence of the friction.

If the cervix remains outside the vulva it becomes deeply congested and it is very likely to become ulcerated. Bleeding may occur from the ulcerated surface, as in the case of E. E—. If the cervix remains outside the vulva throughout pregnancy there may be difficulty and danger in labour, as we shall see later. If the body of the uterus remains in the pelvis, either because it is retroverted and incarcerated under the sacral promontory after acute prolapse or because it is held down by the swollen cervix, pressure-symptoms will occur, viz. pain and retention of urine. On the last out-patient day some of you saw a patient who came complaining of inability to pass her water. She had had a catheter passed three or four times. We found that she was four months pregnant, and that although the fundus of the uterus reached a point two to three inches above the symphysis pubis the greatly swollen cervix was outside the vulva. The whole uterus was pressed gently upwards and a cup-and-stem pessary was inserted.

When pressure symptoms occur due to the inability of the body of the uterus to rise out of the pelvis, it is said that abortion follows if the condition is not relieved.

Treatment.

(1) *During the early months.* — The uterus should be pushed up and a soft ring pessary inserted into the vagina. Care must be taken that the fundus is pushed up, otherwise the uterus may be doubled on itself, the cervix going upwards while the fundus goes down into Douglas's pouch. Such a case has been reported, a condition of incarceration of the retroflexed uterus resulting. To avoid the occurrence of this unfortunate accident the body itself should be pushed upwards and forwards, the patient being placed in the knee-chest position if necessary. If there is any difficulty in effecting the reposition of the uterus, the patient should be kept at rest in bed for twenty-four hours or more and then another attempt made. I have not seen a case in which this was necessary.

The ring should be removed by the end of the fourth month of pregnancy. It will usually be found that it is unnecessary to insert it again, but cases occasionally occur in which a ring pessary must be worn for a longer period. As a rule, the cervix soon becomes much diminished in size when it is freed from the "nipping" effect of the vulval orifice. If there is much vaginal discharge the patient should use a douche—*e.g.* of lead lotion, ʒj of liquor plumbi subacetatis to a pint of water. To prevent induction of abortion by the douche she should be cautioned to use the lotion lukewarm, neither too hot nor too cold, and not to raise the douche-can high enough to cause a forcible flow.

If a ring cannot be retained some sort of a cup-and-stem pessary must be used instead.

In one case I performed ventral fixation during the first month of pregnancy. I do not recommend this method of treatment, though in this case it was most successful. A. C—, æt. 23 years, came to the out-patient department in July, 1904, complaining of prolapse of the uterus, which dated from her first confinement, four years before. She had had a second child, born in 1902. A mass, about the size of an orange, consisting of cervix and anterior and posterior vaginal walls and bladder protruded from the vulva. I decided to perform anterior and posterior colporrhaphy and ventral fixation. She was sent for a few weeks later, when there was a vacant bed, and I proceeded to operate. As the prolapsed mass was rather bluish in colour, I asked when her last period was, and was told that it had finished just three weeks ago. I performed anterior colporraphy, and posterior colpo-perineorrhaphy, advancing the perineum without removal of much tissue, and then opened the abdomen. The cause of the congestion of the cervix and vulva was then evident: the uterus was two or three weeks pregnant. I attached the anterior wall of the uterus to the anterior abdominal wall by a buried catgut stitch which passed through peritoneum and the recti muscles, and one silkworm-gut stitch, which was removed on the tenth day. The patient had some

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pain during pregnancy, but not of great severity. Labour was normal, a large child being born four and a half hours after the onset of labour pains. I saw the patient in May, 1906. The uterus was in the normal position, the cervix normal in every way. The cystocele had recurred, but a ring pessary kept it up well.

(2) *During the later months*, when the cervix remains outside on account of elongation of either the vaginal or supra-vaginal portion, after the end of the third or fourth month.

The uterus should be pushed gently upwards, care being taken as before to see that the fundus goes up. If the cervix is very much swollen rest in bed may be necessary before reposition can be effected.

A ring pessary may be successful in keeping the cervix inside the vagina, but in many cases the vulval orifice is too large, and some sort of cup-and-stem pessary, with strings attached to a waist-belt, must be used. Sometimes an ordinary cup-and-stem pessary, even one of the porcelain ones, with rounded edges, used in the out-patient department, causes pain and bleeding from the cervix. In three or four of these cases I have found that a pessary consisting of a hollow rubber ball, a little smaller than a lawn tennis ball, mounted on a stem with strings attached, answers the purpose exceedingly well and causes no pain or bleeding. It is inflated by means of a syringe after being inserted into the vagina. Like a cup-and-stem pessary, it is taken out at night and put in again in the morning.

E. E— came to Crossman Ward three years ago complaining of pain and discomfort caused by a swelling between her legs. The pain was so great that she had had to stay in bed. She was five months pregnant. The cervix, a large mass with two or three ulcerated patches on it, projected from the vagina. A ring pessary could not be retained in the vagina, and a cup-and-stem pessary caused pain and bleeding if the strings were tied sufficiently tightly to keep up the prolapse. The inflated ball-pessary gave her complete relief, the cervix soon went down to its normal size, and the ulcers healed. She had an easy labour and soon became pregnant again. Profiting by her previous experience, she began to use a cup-and-stem pessary as soon as the symptoms due to prolapse recurred, and she passed through her pregnancy without any discomfort or pain.

A patient with almost exactly the same history was recently confined in Marie Celeste Ward. She first tried a very large ring pessary, which caused pain. Next, she tried a cup-and-stem pessary, which caused pain and bleeding. When she came to the hospital she was given an inflated ball pessary, and this relieved her symptoms.

In some cases it may be necessary to keep the patient in bed for a few days with tampons soaked in a lead or alum solution, to allow the œdema and tenderness to diminish before any sort of pessary is used.

In several cases that have been reported the enlarged cervix has been amputated, sometimes without causing interruption of the pregnancy. In many of the cases, however, abortion has followed. I should not recommend this treatment.

Difficulties and Dangers in Labour.

If the cervix remains outside the vulva throughout pregnancy it becomes not only œdematous, but very firm and tough, and may cause difficulty in labour. Whitridge Williams in his text-book on obstetrics says that as a rule the cervix becomes contracted when labour sets in, though in rare cases it may continue to protrude from the vulva and become markedly œdematous and cause distocia (*i. e.* difficulty in labour), rendering multiple incisions necessary.

I have only seen five cases in which the cervix was outside the vulva at the beginning of labour. In none of these was there any tendency to drawing up of the cervix.

The thickened cervix dilates very slowly. I saw one case, not in this country, in which no assistance was given, and in spite of good pains the labour, in a multipara, lasted over thirty-six hours.

There is a certain amount of danger of rupture of the lower uterine segment of the uterus on account of the mechanical obstruction to delivery offered by the thickened cervix, and there is danger of sloughing of the cervix due to the prolonged pressure to which it is subjected.

I have not been able to find any notice taken of another very real danger to which I want to direct your attention, viz. the danger of septic infection on account of the sloughing condition of the ulcerated cervix.

If the patient is seen some days before the onset

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of labour, or even a few hours before rupture of the membranes, this danger ought to be able to be averted, but that it is a real danger the following two cases will show.

R. F—, a foreign Jewess, æt. 30 years, came to the out-patient department in February, 1904. She was eight months pregnant. The cervix protruded from the vulva, forming a mass nearly as large as one's fist, and could not be pushed up. I advised her to come into the hospital at once so that the cervix could be cleaned up and made as aseptic as possible before labour began, pointing out to her that otherwise there was a danger of infection. She refused to come in at once, but said that she would come in as soon as labour began. A few days later she came back in labour, having pains every ten minutes. The cervix, which was still outside the vulva and ulcerated superficially, was softer than before. It was cleaned up as thoroughly as possible. When the membranes ruptured the head advanced to within two inches of the external os. Five hours after rupture of the membranes the pains became strong and frequent and the head was born suddenly about an hour later. A tear four or five inches long was found in the posterior part of the cervix. The child, which weighed five and three quarter pounds, was alive and did well. The tear in the cervix was closed by catgut sutures. From the third to the tenth day the temperature varied from 98° F. in the morning to 103° F. at night. For the next fortnight the temperature varied between 98° and 101° F. After this the temperature remained normal.

If I had been in the hospital at the time, I should have either dilated or incised the cervix after the premature rupture of the membranes. Two or three small incisions would probably have prevented the large tear. Bossi's many-pronged metal dilators would be useful in such a case.

In the next case the uterus became infected early in labour.

R. S—, æt 32 years, had had ventral fixation performed by another operator about a year before I saw her, on account of prolapse. When I saw her in consultation she was nine months pregnant. The cervix, which was much ulcerated and smelt horribly, protruded three inches outside the vulva, as it had done for some weeks. Her temperature was 102° F. The cervical canal admitted two fingers. Labour pains had begun forty-eight hours before and the membranes had ruptured thirty-six hours before. The fœtal heart could not be heard. The small amount of discharge which was coming away from the uterus was very offensive. I sent her into hospital at once and the resident accoucheur attempted to dilate the tough, firm cervix by pulling on a Champetier's bag and by stretching it with his fingers. As these attempts were unsuccessful I made deep lateral incisions and delivered with forceps. The fœtus stank most horribly. The uterus was washed out with lysol solution. In the evening the patient had a rigor, the temperature rising to 106° F. The next day the temperature was 104° F. and the pulse 120 to 140. As it seemed certain that the uterus was infected and that the patient had severe septicæmia I performed vaginal hysterectomy, hoping that the infection might be still limited to the uterus. The operation was not difficult, the uterus being so soft that it could be brought through the pelvis easily. The total length of the uterus was eleven inches, the cervix measuring four and a half. The cervix was gangrenous round the internal os. The patient stood the operation well, and I was at first hopeful about the final result, but she died of heart failure from septicæmia on the fourth day, in spite of anti-streptococcic serum, etc. There was no peritonitis. I wish that I had removed the uterus immediately after the delivery of the dead fœtus, but at that time I could not say that she had septicæmia.

Such cases as this are probably very rare; they show the importance of treatment before the commencement of labour.

July 16th, 1906.

We hear from Mr. J. C. Inglis, General Manager of the Great Western Railway, that the new direct route between London and the West of England *viâ* Westbury has been opened, and the train service between London and Weymouth, Barnstable, Ilfracombe, Exeter, Plymouth, North and South Devon, and Cornwall has been greatly improved and accelerated. A new through train now runs between Paddington and the London, Brighton and South Coast Company's system in direct connection at Paddington with express trains from and to all parts of the Great Western Company's system. The new route to Ireland *viâ* Fishguard and Rosslare will be opened in August.

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A LECTURE

ON

INFANTILE PARALYSIS.

By GEO. F. STILL, M.A., M.D., F.R.C.P.,

Physician for Diseases of Children, King's College Hospital; Assistant Physician to the Hospital for Sick Children, Great Ormond Street; Professor of Diseases of Children, King's College, London.

GENTLEMEN,—Of all forms of paralysis in childhood infantile paralysis or acute anterior poliomyelitis is the most frequent. And yet, familiar though this disease may be, it is constantly raising difficult practical questions for the clinician, whilst for the pathologist it remains in certain respects an entirely unsolved riddle.

The consideration of its clinical features has important bearings upon the pathological obscurities of this disease, and as a clear mental picture of the morbid anatomy of infantile paralysis is essential to the proper understanding of the questions at issue let me briefly put before you at the outset the main facts of its morbid anatomy as far as they are known. It may be taken for granted now that the clinical phenomena—the paralysis, the muscular wasting, the arrested growth, and the coldness and blueness in the affected limbs—are all due to a primary lesion in the spinal cord, with the reservation that in certain cases peripheral nerve lesions may be associated with the spinal lesion, and that possibly in rare cases a clinical picture hardly, if at all, distinguishable from that of poliomyelitis may result from a primary peripheral nerve affection. To the spinal cord we must look for the characteristic changes, and it is little use seeking to determine the character of the initial lesion or its cause from the changes found in the spinal cord months after the onset of the disease: the shrinkage of the anterior horns, the disappearance or degenerated state of nerve-cells in the anterior cornua, the

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diminished size of the anterior roots, the ascending degeneration which may occur, these tell us little or nothing of the beginning of the disease. It is from the examination of cases which have died during the first few days of the disease that we may hope, sooner or later, to determine its cause; at present but few such early examinations have been recorded. My colleague, Dr. Batten, has recorded a carefully studied case which died thirteen days after the onset, and I quote his description of the changes found:

"The vessels in the grey matter are thrombosed and surrounded by perivascular exudation; there are scattered hæmorrhages throughout the grey matter and exudation of small cells. The congestion is almost limited to the ventral portion of the cord."

Now, upon the interpretation of these early changes there exists considerable doubt. Is the thrombosis the primary lesion, or is there first an inflammation of which the thrombosis is merely a part? In either case is the primary lesion the result of invasion by some micro-organism? and if so, is the infection one peculiar to this disease? or, on the other hand, may the lesion result from many different causes, infective and otherwise? These are the questions raised by the morbid anatomy of infantile paralysis, and if it be not possible to give a definite answer to any of them, yet we may at least obtain some hints towards their solution from a consideration of the clinical facts of the disease.

Boys would seem to be rather more liable than girls. My own figures show in 87 cases 50 boys to 37 girls.

The age incidence is of some interest; the disease may occur even in adults, but it usually begins before the end of the third year, and is far more frequent during the second year of life than at any other period. Out of 85 cases in which I have noted this point 70 began within the first three years of life, and there is a very striking maximum incidence in the second year; 33 began between the ages of one and two years, whereas only 15 began in the first year of life. The earliest age of onset among my cases was eight weeks. I draw your attention to the special incidence upon the second year of life because it contrasts with the age incidence of infantile hemiplegia, which has its onset most often in the first year.

With reference to the age incidence I would remind you also, but without laying any great stress upon the fact, that such undoubtedly infective diseases as suppurative, posterior basic or cerebro-spinal, and tubercular meningitis have their maximum incidence during the first three years of life, so that it might be suggested that the central nervous system is specially liable to infective disease at this period.

More important is the seasonal incidence; in 45 out of 68 cases in which the month of onset could be determined the disease began between the beginning of June and the end of September; and in 30 out of the 68 cases the onset was either in August or September. This incidence during a particular season strongly suggests an infective character; but undue stress must not be laid upon it, for it must be remembered that diseases which I suppose no one imagines to be infective have sometimes a very striking seasonal incidence—for example, spasmus nutans; and it is quite possible that light, temperature, and barometric pressure may influence the seasonal incidence of a disease apart from any infective agent.

A further point which might be urged in favour of an infective origin for infantile paralysis is the relation of this disease to specific fevers; amongst my notes of 87 cases there are 7 in which the paralysis supervened during the acute stage of a specific fever or during convalescence therefrom: (1) fourteen days after the appearance of measles rash; (2) four weeks after measles; (3) eight days after onset of scarlet fever; (4) while in hospital convalescing from scarlet fever; (5) during typhoid; (6) during whooping-cough: (7) eight days after vaccination, paralysis of vaccinated arm. The tendency for one specific fever to follow another is often apparent in children who within a few months will pass through two or three specific fevers, and is, I think, a reasonable ground for assuming that if any particular disease tends to follow specially upon specific fevers the disease in question is likely itself to be of specific infective nature. But there is another possible explanation; thrombosis may certainly result from typhoid fever, and it seems likely enough that after other acute illnesses degenerative changes in vessel-walls and feeble circulation, perhaps with dilatation of the heart, may favour the occurrence of thrombosis apart from any specific infection peculiar to infantile paralysis;

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on this view the relation to specific fevers might be held to support the idea of a primary thrombosis as the cause of the spinal cord condition. It is noteworthy that infantile paralysis follows directly, not only the specific fevers, but also acute diarrhœa; in three of my cases acute diarrhœa was followed after twenty-four hours' duration by infantile paralysis, and in another case paralysis was found after the child had been ill three weeks with diarrhœa. A primary vascular lesion would accord also well with the cases in which traumatism has seemed to be the determining factor. Parents are apt enough to attribute illnesses to blows and falls the occurrence of which just before the illness may be a mere coincidence; but it is as easy to attach too little importance to them as too much, and even if a disease be undoubtedly of infective character it may still be true that a blow or fall may produce either at the part struck a *locus minoris resistentiæ*, or by its generally depressing effect a general vulnerability of tissues. For instance, a boy æt. 5½ years went to school apparently well; whilst there he was hit a painful blow in the back by another boy; in the afternoon shortly after the blow he fell whenever he tried to walk, both legs were paralysed; the right recovered gradually, the left remained paralysed. In two other cases the onset of infantile paralysis was three days after a fall downstairs, but in both the onset was accompanied by fever and in one by vomiting, as is common in cases where there is no history of traumatism.

The strongest argument in favour of an infective origin in the absence of bacteriological proof is the occurrence of epidemics of infantile paralysis, a fact which seems also strongly to support the view that the disease is due to some specific infection. Whilst, however, I draw your attention to this almost conclusive proof of an infective origin, I would remind you that in most of these epidemics there have been certain features very unlike those we are familiar with in the ordinary sporadic infantile paralysis. In the first place, the mortality has been widely different from that of the sporadic disease, which is very rarely fatal. In the epidemic recorded by Caverley at Rutland, Vermont, U.S.A., there were 18 deaths out of 126 cases—*i. e.*, nearly 15 per cent. Again, in an epidemic in France there were 3 deaths among 13 cases. Moreover, it is remarkable that in some of these epidemics some cases have shown symptoms so like cerebro-spinal meningitis that great doubt has arisen as to their nature, and in the most recent epidemic in Australia one view regarded the epidemic as including two distinct diseases—acute anterior poliomyelitis and cerebro-spinal meningitis. In the Rutland epidemic horses, dogs, and fowls were affected, and in a horse the necropsy showed that there was definite meningitis. In some of the epidemics some of the cases have resembled rather a polyneuritis, and this also has been demonstrated at autopsy. Lastly, I would point out that complete recovery, which is so rare in ordinary sporadic infantile paralysis, has been quite a common occurrence in some of the epidemics. Caverley reported 50 complete recoveries in 126 cases; Altmann, in an Australian epidemic, reported 4 complete recoveries in 18 cases.

I mention these facts, not to throw any doubt upon the occurrence of epidemic infantile paralysis, but only to suggest that until we have some bacteriological or other test of the specificity of infantile paralysis the exact relationship of the recorded epidemics to the sporadic disease which we call infantile paralysis remains uncertain.

Onset and symptoms.—The clinical course of infantile paralysis gives but conflicting evidence on the problems of its pathology. Most striking, to my mind, are the remarkable differences in the mode of onset, which may be classified in three groups:

(1) An acute illness, more or less severe, lasting a few days, with pyrexia and drowsiness, and perhaps with vomiting, headache, constipation, and some pain in the back or limbs, is found to have left the child paralysed in one or more limbs. *E. g.* Alice S—, æt. 18 months, on October 9th was restless and seemed vaguely ill; on October 10th she vomited three times and was feverish; October 11th she seemed less ill, but on October 12th she was noticed to be unable to move her limbs or head, and as she improved she remained paralysed in the left arm and leg.

This is the commonest mode of onset and would fit in well with the idea of an infective disease.

(2) An insidious onset which perhaps cannot be dated at all; the first thing noticed is weakness, and this perhaps only when there is already considerable wasting. *E. g.* Charles H—, æt. 2½ years, came with flaccid paralysis of the left leg. The onset was entirely unnoticed, and it was only when

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he should have walked at sixteen months that the weakness of the left leg attracted attention. In some of these cases one is assured the weakness has come on gradually, but it seems likely that it is the wasting rather than the weakness which has gradually increased and has given rise to this statement.

(3) Sudden, perhaps instantaneous, onset. *E.g.* Violet S—, æt. 2 years and 4 months, was running about at play; she fell down, and on being picked up was found to be paralysed in the right leg. I have seen another case with exactly similar history, where a little girl running across a room suddenly dropped down and was found to be paralysed in the legs. In both these cases the subsequent condition seen by me was that of typical infantile paralysis. Such cases suggest very strongly the occurrence of some vascular lesion, obstruction, or hæmorrhage. In this group, perhaps, should be included the not uncommon cases of which the following is an instance: Willie S—, æt. 3 years, went to bed apparently perfectly well; in the morning he was found to be paralysed in the right leg, though otherwise showing no sign of illness; the paralysis remained permanent.

In connection with the onset of the disease I would draw your attention specially to the evidence of widespread disturbance which is noticeable in some cases at this stage. Not only is there fever, with malaise, but in 7 out of 87 cases I have noted convulsions at the onset; and in one case a boy, æt. 4½ years, whose illness had begun with a convulsion, followed by vomiting, headache, and drowsiness, fell into a semi-comatose condition, his face became paralysed on the left side, he passed his urine and fæces under him, and after a few days lost power in the left shoulder muscles and the right leg. All these symptoms disappeared within three weeks from the onset, except the paralysis of the right leg, which persisted with wasting of muscles and "reaction of degeneration."

In such a case it seems evident that there is something more than a mere local affection of the spinal cord, thrombotic or otherwise; there is a profound constitutional disturbance suggestive of some acute infective disorder. The facial palsy in this case was of interest as showing that parts at least as high as the pons may be affected. My colleague, Dr. F. E. Batten, has published a case in which microscopic examination proved that the facial paralysis was the result of congestion which "had destroyed the nucleus of the seventh nerve"; and if the disease may produce inflammation of the grey matter at this level there seems to be no reason why it should not produce it also in the grey matter of the cortex. On this view the polioencephalitis which is supposed by some to be the usual primary lesion in "infantile hemiplegia" may be of a nature identical with the poliomyelitis which is the usual lesion in "infantile paralysis."

Similarly, "infantile paralysis affecting the brain" may be the explanation of the widely spread polioencephalitis which probably underlies some of those cases in which, after a sudden onset of convulsions with loss of consciousness and pyrexia, a child is left idiotic with general spasticity. But until we have bacteriological evidence by which we may test the identity of such conditions it would be premature to state that these cortical lesions are etiologically identical with infantile paralysis.

It seems clear that for some reason the part of the spinal cord remote from the brain is affected most often and most severely. My own figures showed in 85 cases:

Left lower limb paralysed in . .	27
Right ,, ,, . .	19
Both ,, ,, . .	13
Both lower and both upper limbs .	6
,, and right ,, .	2
,, and left ,, .	1
Right lower and left ,, .	2
Left ,, ,, ,, .	1
Both upper limbs	2
Left upper limb	5
Right ,, ...	7

It will be seen that the lower limbs, one or both, were the only part affected in 59 out of 85 cases: that the lower limbs were affected together with the upper in 12 more cases, and that the upper limbs alone were affected only in 14 cases.

These figures refer to the permanent paralyses, but it must be remembered that during the few days or weeks following the onset of the disease there is often much more extensive paralysis, which clears up, leaving, it may be, only one limb or one group of muscles affected.

I have not referred in these figures to paralysis of trunk muscles which, though much less common than that of limbs, is sometimes a marked feature; twice, at least, I have seen the diaphragm para-

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lysed, and cases have been recorded in which the muscles of the abdominal wall have been paralysed on one side or both, so that the abdomen was puffed out in an extraordinary manner when the child cried or coughed. The spinal muscles were affected in several of my cases, giving rise to more or less lateral curvature; the following case illustrated both the extensive recovery from initial paralysis and the persistence of trunk affection. Winifred P—, at the age of eighteen months, became feverish and ailing one day in August; after three days it was found that she was weak in all her limbs, but especially in the right leg and left arm. After a few weeks the limbs had gradually recovered completely, but months afterwards there was severe lateral curvature left as a result of affection of spinal muscles.

The results of infantile paralysis are not limited to the loss of muscular power; in many cases there arise gradually secondary deformities which add in no small degree to the disability already present; such deformities, which include several varieties of talipes, claw-hand, etc., are dependent chiefly on unopposed action of the unparalysed muscles, but partly, for instance in talipes valgus, on stretching of ligaments owing to absence of muscular support.

It is sometimes stated that there is no affection of sensation in infantile paralysis, and this no doubt is true for the disease in its chronic stage, but at the onset I have often found marked tenderness in the affected limbs, so that the child cries whenever the limbs are touched; in some cases also there is pain in the affected limbs at this stage. Diminution of sensation is much rarer than tenderness; it has been observed during the first few days of the disease.

As in a case I have quoted, incontinence of urine and fæces, or retention of urine, sometimes occurs at the onset, but although these irregularities may indicate affection of the sphincters in some cases, they are probably more often due to the negligence in these matters which is common in children with any acute illness or disablement; I have never known any sphincter affection to persist more than a fortnight after the onset.

The tendon-jerks are rapidly lost in the affected limbs, but I would remind you that this loss depends entirely upon the distribution of the paralysis; there may be extensive paralysis in the legs with no loss of knee-jerks, so long as part of the quadriceps extensor, particularly the vastus internus, is intact. Out of 64 cases in which the lower limb was affected I have noted that the knee-jerk was present in the paralysed leg in no less than 24 cases. The plantar reflex has shown extensor response in some of my cases, signifying, probably, not interruption of pyramidal tracts, but simply weakness of flexor muscles, so that the unparalysed extensors predominate.

Diagnosis.—When the limb is already wasted, cold and blue, with loss of tendon-jerks and with flaccid paralysis, the diagnosis is usually easy enough, but at the onset a diagnosis may be impossible for several days, and the febrile illness, with tenderness or pains in limbs and back, has been mistaken, naturally enough, for all sorts of febrile ailments; acute rheumatism it was supposed to be in a little girl, who, with some fever, complained of tenderness about one shoulder and inability to move it; and this, perhaps, is one of the commonest errors. Influenza the illness was called in another case, while in the case where facial paralysis was associated with stupor and fever before the paralysis of limbs appeared, a confident diagnosis of meningitis was made. Even when extensive paralysis of limbs is evident, it must be remembered that there are other conditions beside anterior poliomyelitis which occasionally cause such symptoms in children. I have seen acute myelitis paralyse all four limbs in a child of six years, but the occurrence of anæsthesia, with retention of urine, cystitis, and a tendency to bed-sores, all pointed to this diffuse inflammation of the cord. Neuritis also may hardly be distinguishable from poliomyelitis, unless the occurrence of prolonged tenderness and much pain in the limbs may be taken as indicating peripheral nerve-lesion rather than affection of spinal cord, but in some of the epidemics of supposed infantile paralysis neuritis was found to be associated with the cord-lesion, and it may be that the specific *materies morbi*, if such there be, of infantile paralysis can act upon nerves or spinal cord indifferently; certainly there are cases in which pain and tenderness, associated with very extensive paralysis and a very slow recovery in almost all the affected parts after many weeks or months, strongly suggest a polyneuritis rather than a spinal cord lesion, but the distinction may be impossible.

Prognosis.—There are few diseases which within

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so few hours or days may so completely mar a life by permanent and hopeless crippling as does infantile paralysis. And yet, disastrous though its results may be, it is a disease which, at any rate in its ordinary sporadic form, very rarely endangers life. When infantile paralysis kills it is almost always during the first week or two of the disease, and death is due to paralysis of respiratory muscles from affection of the upper part of the cord, particularly of that part of the cervical cord which supplies the diaphragm. But it is possible that more deaths may be due to this disease than are commonly attributed to it; for if it affects the cervical cord there seems to be no reason why it should not affect the medulla, and it may be that some of the sudden and unexplained deaths in children may be due to infantile paralysis affecting the medulla, and so damaging the respiratory and cardiac centres that death occurs before any paralysis of limbs has appeared.

Does complete recovery ever occur in infantile paralysis? It is usual for some of the limbs or muscles originally paralysed to recover completely within a few days or weeks after the onset, and if the lesion in the cord may thus subside in one part without permanently damaging its functions there seems to be no *a priori* improbability that all the parts affected may sometimes recover. There is evidence, as already mentioned, that this has happened in some of the epidemic cases, but naturally it is difficult to prove any sporadic instance. A child was brought to the Children's Hospital with infantile paralysis of one limb; it was stated that another child in the family had become paralysed similarly within ten days after the onset in this child, but had completely recovered.

The possibility of complete recovery is strongly suggested also by the not uncommon cases in which the weakness left some months after the disease is so slight that it might be overlooked were it not for the wasting and shortening of the limb, which is more evident than the weakness.

If complete recovery occurs it is certainly to be looked for only in the first few weeks after the onset; later, the only question is as to the degree of improvement possible. And here I would emphasise two points: (1) Do not take too gloomy a view because the paralysis, say three months after the onset, is extensive in distribution and the muscles flabby and wasted. No doubt a complete loss of reaction to faradism and galvanism makes it improbable that the muscle will recover, and a "reaction of degeneration" is only less discouraging, but I would strongly advise you not to base your prognosis merely on electrical reactions. The origin of nerve-supply of a muscle extends probably through two or three segments of the spinal cord, and it is always possible that, though the main sources may be irreparably damaged, some may be so little affected that some small portion of the muscle may retain its function and, by compensatory overgrowth, restore to some extent the lost power. Moreover, where some muscles have escaped the child gradually learns to adapt its remaining powers to the altered mechanical conditions. The power of walking may thus improve steadily for years, not because there is any real recovery in affected muscles, but because the child learns to use to the best advantage those which remain, and the extra work thrown upon these muscles, or surviving portions of muscles, increases them in size and power. (2) Do not take too bright a view, because the paralysis is very slight and limited in extent. It is a striking feature in some cases of infantile paralysis that the arrested growth in the bones of the limb may be altogether out of proportion to the paralysis. A boy who, at the age of three and three quarter years, had infantile paralysis in the left leg, had so little weakness at the age of ten years that all movements seemed to be good, but, nevertheless, he limped badly in walking, for the affected leg was two inches shorter than the other.

Treatment.—It is conceivable that in the first few days of the disease good may be done by measures directed to reducing the hyperæmia of the spinal cord, particularly by application of ice-bags over the affected portion or by vigorous counter-irritation, *e.g.* with mustard plasters. I have used and seen used various measures of this kind, but as some of the initial paralysis almost always recovers whether any local treatment has been used or not, it is difficult to determine whether the amount of residual and permanent paralysis is influenced by such applications. I have also used ergot internally. A child of eighteen months will take ten minims of the liquid extract, and a child of five will take thirty minims three or four times a day; aconite and

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eserine have also been used; but here also the treatment rests on theory, not on demonstrable results. Examination has shown that in the affected portion of the cord some inflammatory exudation persists for several weeks. It seems reasonable, therefore, to give iodides for a considerable period, perhaps two or three months, in the hope of promoting absorption and so relieving the pressure which no doubt hinders repair.

Apart from these measures of theoretical utility can we influence the condition in the spinal cord? One would fancy, to hear the extravagant claims which have been made sometimes for electrical treatment or massage of the paralysed limbs, that such treatment had some influence upon the seat of the disease; but surely one might as well water the branches of a dead tree in the hope of reviving its root as stimulate the muscles with the idea of restoring the destroyed cells in the spinal cord. What we can do—and the only thing we can do by such treatment—is to promote the nutrition of those muscles or parts of muscles whose nerve-supply has escaped destruction, and so to raise their working capacity to its utmost. There is no doubt that just as the athlete by continual use of one muscle or set of muscles increases it both in size and power, so by frequently repeated stimulation, whether by electricity or by specially adapted exercises, and by flushing the muscle with blood, as is done by massage, we can increase both in size and power those muscles or parts of muscles whose representation in the cord has not been destroyed. With this purpose electricity, massage, and carefully planned exercises are all of value after the first two months of the disease, but I doubt whether electricity has so great a superiority over massage as to commend its use for those children who never seem to lose their dread of it, and to whom its daily application means daily misery. But no treatment is of much avail which is not capable of home application, for it requires to be used, not once or twice a week, but twice or thrice daily, and this is seldom practicable unless the parents or nurse can carry it out themselves. While I am convinced that in expert hands the application of galvanism is of considerable value, I am not at all sure that its random application by mother or nurse does much good, and as they can be taught to carry out efficient massage with excellent results, I am inclined to recommend massage as the more generally useful treatment. Not less valuable than either electricity or massage are carefully adapted exercises for cases in which a limb is only partially disabled; but they must be planned with care and discretion, for it is obviously possible to do harm rather than good by encouraging the unbalanced action of muscles which are already tending to cause deformity by their unopposed power of contraction. Where any voluntary contraction of a weakened muscle is possible, or where by extra development of unparalysed muscles some compensatory advantage may be gained, the surest method of increasing power in these muscles is by exercising them frequently.

I have said nothing of the surgical measures which are occasionally advisable in infantile paralysis, tenotomy, fixation of joints by "arthrodesis," and the recent methods of tendon-grafting; each has proved its value in particular cases, but their consideration does not fall within my province. On one matter, however, which is perhaps more surgical than medical, I venture to speak. Is it wise to encase a child's leg in a rigid apparatus which effectually prevents any exercise of the very muscles which being weak require to be used? I am well aware that there is a Charybdis as well as a Scylla to be avoided, and that some deformities—*e.g.* talipes valgus or genu valgum—may be aggravated in some cases by walking without artificial support; but none the less I think that sometimes a very simple support, perhaps even a flat-foot pad or some stiffening in the side of the boot, assisting but little the action of the weakened muscles, might give the limb a better chance of improvement than does the elaborate and cumbersome instrument which has been worn. Certainly if such apparatus must be used, it should be left off for some hours daily when the child is not walking, and in these intervals the muscles should be improved by massage and exercise. Lastly, I would point out that while in all cases there comes a time when no further improvement is possible, this may not be until two or three years or even more have elapsed since the onset; recovery from paralysis there is probably little or none after the first few months of the disease; but improvement, in the sense of increased ability to perform certain actions, there certainly is for much longer, and this, as I have said, depends upon extra development of the muscles or parts of muscles which have escaped.

July 23rd, 1906.

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CIRRHOSIS OF THE LIVER.*

By GUTHRIE RANKIN, M.D., M.R.C.P.,
Physician to the Dreadnought and Royal Waterloo Hospitals.

GENTLEMEN,—In the wards of this hospital you will find frequent opportunities for making yourselves familiar with almost every variety of hepatic disease. I am able to-day to bring under your notice two cases illustrative of leading types of cirrhosis. Before proceeding to their investigation I may remind you that cirrhosis of the liver is a pathological condition which owes its incidence almost entirely to three causes—alcohol, syphilis, and malaria. It is not therefore suprising that in a hospital devoted to the treatment of seamen, among whom as a class these etiological antecedents are prevalent, we should meet constantly with examples of this form of disease. It is true that cirrhotic changes in the hepatic tissues are encountered, from time to time, under conditions which exclude each and all of these three great predisposing factors, but they constitute such a fractional proportion to the sum total of cases of the disorder that, for the purposes of our present inquiry, they may be set on one side as comparatively unimportant.

Of the three causes which I have mentioned, alcohol is responsible, and may be alone responsible, for the majority of cases, but alcoholism is frequently associated with syphilis, or malaria, or both, especially among the patients who seek the asylum of the Seamen's Hospital Society.

In both of the cases before us there is a history of syphilis, and in one an indefinite narrative of malaria, but, what is of more importance from the diagnostic point of view, there is in both an admitted record of excessive alcoholic indulgence over a long period of time.

It is generally recognised that alcohol is capable of producing two distinctive types of cirrhosis, and in these patients we have probably each variety represented. The most common form of alcoholic cirrhosis is that which produces the "hob-nail" liver originally described by Laennec. In it the newly-formed fibrous tissue is deposited in the larger portal canals and surrounds groups of hepatic lobules, so that it has come to be spoken of as "multilobular." The second type of the disorder is designated "unilobular" because the adventitious tissue is developed in the smaller portal tracts around single lobules; in the newly developed interlobular connective tissue bile-ducts become formed, and from this circumstance Hanot and others promulgated a theory that the origin of the cirrhosis in such cases was biliary. This hypothesis, though not wholly abandoned, has not been conclusively verified by later investigations.

On account of the diminution in size and deformity of the "hob-nail" or "multilobular" liver, it has sometimes been referred to as "atrophic"; and, *per contra*, on account of its increased size the "unilobular" variety has been described as "hypertrophic." These terms are confusing and should be abandoned, especially as "multilobular" cirrhosis is now known to be frequently accompanied by such a deposition of fat that the liver, instead of being diminished, is markedly increased both in size and weight. It is probable that syphilis is never directly responsible either for "multilobular" or "unilobular" cirrhosis; but it produces anatomical changes in the liver which are no less characteristic than those resulting from alcohol. Hereditary syphilis is the direct cause of an interstitial hepatitis which is seen either in stillborn children or in infants who have survived birth for only a few weeks or months. It gives rise to a uniform increase in size of the liver, which is sometimes accompanied by jaundice and moderate ascites, but which may be without clinical signs other than the enlargement. In these cases the new tissue penetrates the lobules and isolates groups of cells, or individual cells, so that it has come to be known as "intra-lobular" or "pericellular" cirrhosis. In acquired syphilis the development of gummata is undoubtedly the starting-point of the cirrhotic changes which ensue. Gummata also occur, though rarely, from hereditary taint. The gumma may occur singly, but more frequently several develop; they are variable in size and may be found anywhere throughout the hepatic boundaries, but they have a special affinity for the neighbourhood of the suspensory ligament at the junction of the right and left lobes. When gummata undergo caseation they become replaced by fibrous tissue; obviously, therefore, the ultimate

* A lecture delivered at the London School of Clinical Medicine.

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result upon the liver must vary according to their number and position. A few superficial scars may be the only vestige they leave behind, but on the other hand, the whole organ may be furrowed and intersected with broad, fibrous bands as the consequence of their presence. We will now ask Dr. Thresher to read to us a short account of these patients' illnesses as recorded in the hospital notes:

CASE 1.—W. P—, a seaman, æt. 48 years, was admitted on May 11th. He complained of cough, palpitation, shortness of breath, and swelling of the legs of three weeks' duration.

There was a history of a similar swelling six months previously, which had gradually disappeared. He had suffered from morning sickness, and admitted having drunk beer to excess for several years.

"*On admission*: He is a fat, flabby man. His face is dusky and puffy; his cheeks are covered with rosaceous acne, and his sclerotics are slightly icteric. His breathing is laboured and accelerated. Both legs are œdematous. His chest is resonant all over; rhonchi and crepitations are abundant throughout both lungs.

"The apex beat is diffuse and cannot be accurately felt, but the right border of cardiac dulness extends half an inch beyond the right sternal margin. Two systolic murmurs can be heard, one at the apex, which is conducted some way into the axilla; and the other, of softer quality, on the right side of the sternum, at its junction with the fifth costal cartilage. Over the pulmonic cartilage the second sound is reduplicated. The pulse is of low tension, irregular, and numbers 132. The liver is enlarged and extends to two and a half inches below the costal arch; its lower edge is well defined and it is little, if at all, tender.

"The spleen cannot be felt. There is no fluid in the abdominal cavity."

On the 22nd of May—that is, eleven days after admission—there is this further note, which is important:

"Temperature has risen this morning to 102·4° F. There is dulness on percussion throughout the right lung, with bronchial breathing, increased T.V.F., and a few scattered moist râles. No tubercle bacilli have, on a first examination, been found in the sputum. The apical murmur has disappeared and the cardiac rhythm is now regular. Over the lower end of the sternum a soft systolic bruit can still be heard. The liver is no longer tender and has diminished somewhat in size; it is still below the costal margin."

CASE 2.—J. H—, a seaman æt. 52 years, was admitted on April 24th. He gave a history of profuse hæmatemesis one week previously. There was a record of his having vomited blood and passed tarry motions on several occasions within the previous five years.

He had suffered from indigestion and occasional vomiting for twelve years, but had not remarked any tendency to morning sickness. He admitted having drank spirits to excess, and mostly raw, since his youth.

"*On admission*: He is a spare man, with a florid face and dilated venules over the malar bones; his tongue is furred and tremulous; his chest is somewhat emphysematous and there is marked prolongation of the expiratory part of the respiratory murmur; the cardiac dulness is diminished; there is no murmur, but the second aortic sound is accentuated. The vessels are thickened and there is distinct increase of arterial tension.

"The upper margin of liver dulness begins at the seventh rib, and the lower margin apparently ends above the costal margin. The lower edge cannot be felt through the abdominal wall even on deep pressure.

"The urine contains neither casts nor albumen, but its density is only 1010. The amount passed in twenty-fou rhours measures forty-five ounces."

Though both cases are labelled cirrhosis of the liver, it will be at once apparent, from the account you have heard, how widely they differ from one another in the symptoms they present.

W. P— has a smooth, firm enlargement of his liver, the lower edge reaching well below the costal arch. When admitted he was suffering from cough and he had a certain degree of cyanosis. On examination it was found that he had a systolic murmur at his mitral area and also over the lower end of the sternum. These murmurs have now disappeared and no trace of abnormality remains other than some impurity of the first sound at the apex.

Doubtless under the influence of portal obstruction, aided probably by some debility of his cardiac muscle from recent alcoholic indulgence, this man, when admitted, was suffering from regurgitation through his tricuspid and mitral orifices, but with

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the improvement which has come to him since he has been under treatment both valves have become competent. The bronchitis, of which there was evidence, has also disappeared. We have heard that he presented marked swelling of his legs, but there was no fluid in the abdomen.

I think we may fairly assume that this œdema, which has now subsided, was cardiac and dependent upon backward pressure. The patient is fat; even in his present improved condition his complexion is "muddy" and his features flabby. He admits freely that he has been a copious drinker, but it is of interest to be told by him that his tipple has been exclusively beer. This fits in with his general appearance and is not inconsistent with the type of cirrhosis from which we believe him to be suffering. His case is probably one of the "unilobular" variety. The increased size of his liver, which we can so readily feel, may be partly the result of fatty changes, but if the cirrhosis were of the "multilobular" type, with which fatty changes are frequently associated, the surface of the enlarged viscus would not be smooth and regular as is the case here. The absence of ascites is notable, and also the fact that, up to the present, there has been no marked jaundice. The former symptom is not common in this variety of the disease, but the latter is, and we may feel confident that unless the progress of his ailment becomes arrested, the skin and other tissues will sooner or later become bile-stained. Already we have evidence of biliary obstruction in the condition of the urine, which is of normal density, contains neither albumen nor sugar, but is, as you see, highly coloured from the presence of an excess of bile. The spleen cannot be felt. Now let me ask you to glance at his temperature chart. You will observe an occasional fugitive pyrexia, which is probably toxæmic, and is not a very uncommon manifestation in gastro-hepatic disorders. But you will further notice that the most recent pyrexial attack, which commenced a fortnight ago, has not subsided, and has assumed an unpleasant up-and-down curve. Within the period of this pyrexial disturbance his cough has increased, and certain new physical signs have declared themselves in his right lung. There is increased vocal and tactile fremitus, and a few small moist râles are scattered throughout the entire area of the lower lobe; the respiratory murmur is bronchial in quality over the base, and becomes almost tubular near the apex of the lobe. This is very suggestive of tuberculous trouble, but so far the bacillus has not been found in the sputum. It will be searched for from time to time should these physical signs continue. Meantime their presence is convenient to our present purpose, because they remind us that tuberculosis is one of the most frequent, and also one of the most fatal, complications of hepatic cirrhosis.

Turning now to our second case, J. H—, we find quite another picture. And the first striking fact which appeals to us is the different aspect of this patient from that of the man whose bedside we have just left. He is spare, fresh-complexioned, and has marked distension of the capillaries over the prominences of each cheek-bone. He is alert and active, but tells us that recently he has been losing flesh, and that over a period of some years his digestion has been impaired. He has been subject to nausea and discomfort from flatulence, with occasional attacks of diarrhœa. Like our other patient, he confesses to a free indulgence in alcohol, but he tells us that with him "hard stuff," meaning thereby whisky or rum, was his weakness, and that he drank it always either neat or diluted with a minimum of water. With a history of spirit-drinking over many years, and persistent gastric catarrh such as we get in this case, it is not to be wondered at that the event which ultimately drove him to hospital was a copious hæmorrhage. He had both hæmatemesis and melæna, and, from his own account, must have lost a considerable quantity of blood. In the early stages of portal obstruction a condition of passive hyperæmia becomes established in the stomach and intestines, upon which depends the imperfect digestion, morning nausea, diarrhœa, and other evidences of gastro-intestinal catarrh which are always more or less prominent early phenomena in these cases. Gradually this hyperæmia becomes more pronounced and eventually Nature endeavours to relieve the obstruction in the portal circulation by establishing an anastomosis between it and the systemic circulation. This she accomplishes especially in three situations: first, at the lower end of the œsophagus; second, in the rectum; and third, at the umbilicus. When anastomosis is thus fully developed the systemic veins which are called in to relieve the portal obstruction become enlarged and often varicose. From the relief

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which they afford to pressure within the portal system they lessen the risk of ascites, but their own overcharged condition renders them specially liable to rupture. This patient is an instance in point: his hæmorrhage proceeded from dilated veins situated either at the lower end of the œsophagus or in the cardiac extremity of the stomach, and so far he has escaped ascites. When the veins round the umbilicus become dilated from intercommunication through parumbilical veins between the portal and epigastric systems, they may assume such a size as to deserve the name of "caput medusæ," which Osler and others have applied to this condition. More frequently, however, the anastomosis is evidenced only by abnormally prominent veins running from the umbilicus upwards towards the thorax and downwards towards the groins. The occurrence of piles is the tangible manifestation of anastomosis between the hæmorrhoidal and inferior mesenteric vessels, and there can be little doubt that in early stages of cirrhosis copious hæmorrhage from the hæmorrhoidal veins is a provision for safety. When we come to examine this patient we find the facts related in the report you have heard read fully confirmed. His liver-dulness is markedly diminished; there is slight enlargement of his spleen; his arteries are thickened and tortuous, and manifest a moderate degree of plus-tension; his heart is normal, but the second aortic sound is accentuated; the urine is of average amount, pale in colour, and contains no albumen, but it has an ominously persistent low density of from 1010 to 1015. Since admission there has been no recurrence of bleeding, and the general condition has so much improved that the man is impatient to resume his work. Please observe that there has never been any suspicion of jaundice in this patient, and now, as you look at him, I think you will agree with me that his complexion is remarkable for its freshness and his eyes for their clearness and brilliancy. Also, as I have already pointed out, he has hitherto had no evidence of ascites, but in this case that is the danger ahead, and when it arrives the patient will have entered upon the terminal stage of his illness. If a patient in this condition never touches alcohol again he may escape any greater penalty than a hæmorrhage such as this man has already experienced; but if he is tempted to resume his old habits, the day will inevitably come when his abdomen will fill with fluid; his nutrition will become so impaired that he will rapidly emaciate; his bright complexion will become replaced by a sallow, cachectic hue; he will become mildly delirious, and show other evidences of toxæmic poisoning; and unless carried off by an intercurrent hæmorrhage or a low form of pneumonia, he will ultimately die from asthenia.

The "multilobular" and "unilobular" varieties of hepatic cirrhosis may be differentiated as follows:

Unilobular.	*Multilobular.*
1. Patient usually fat, bloated, and inert.	Patient usually spare, alert, and bright complexioned.
2. Advice first sought either on account of jaundice or of some sudden disorder such as bronchitis or cardiac dilatation.	Advice first sought either on account of hæmatemesis or of increasing enlargement of the abdomen.
3. A history of long-standing gastric catarrh and of many years' addiction to malt liquor in excessive quantities.	A history of gastric catarrh, following long-continued indulgence in ardent spirits.
4. Smooth, firm, painless enlargement of the liver.	Diminution of liver-dulness, or if organ is enlarged, recognisable irregularity of surface.
5. Normal spleen.	Enlargement of spleen.
6. No dilatation of systemic veins.	Dilatation of epigastric, hæmorrhoidal or œsophageal veins common.
7. Jaundice, which may ultimately become deep and persistent.	No real jaundice; slight icterus in late stages.
8. No ascites; sometimes œdema from cardiac failure.	Ascites, as an ultimate development, in the majority of cases.

In alcoholic cirrhosis the complications most to be feared are tuberculosis and interstitial nephritis. In the case of W. P— there is recent evidence in his right lung that the first of these conditions has set in; in that of J. H—, the specific gravity of the water is highly suggestive of the prospective advent of the second. But apart from the occurence of complications, the prognosis is never good. Once established, hepatic cirrhosis of alcoholic origin is mostly fatal, principally because the disease is generally far advanced before it is subjected to proper treatment and because the habits which have produced it have become so deep rooted that it is almost impossible to hope for their correction.

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The occurrence of ascites in the "multilobular" variety of the disease is an unfavourable symptom, and probably most of the cases which are reported to have recovered after its development have really been patients in whom syphilis has played a more prominent causative part than alcohol. Once sclerotic changes are established, the damage done cannot be repaired, but the progress of the disease may be arrested if it is dealt with at a very early stage before the new fibrous tissue is present in great amount, and if for the rest of life alcohol is persistently shunned.

Time will not allow of any detailed reference to cirrhosis of malarial or syphilitic origin. The former, which is rare, may assume either the multilobular or the unilobular type, and is liable to become accompanied by lardaceous changes which are unknown in alcoholic cases. The latter possesses distinctive characteristics which have already been referred to, and which clearly differentiate it from the corresponding alcoholic affection.

As regards treatment there is not much to be said. When the evidence points to a syphilitic causation iodide of potassium and mercury should be administered; where malaria may be in part responsible, quinine combined with suitable tonics and residence in a non-malarial country are the clear indications. In all varieties of cirrhosis it is safe to lay down as a first principle of treatment the uncompromising prohibition of alcohol in any form. The diet should be simple and nutritious. Meat should be taken in great moderation, and the more milk can be depended upon for supplying the daily requirements the better. For a time the patient ought to be kept completely at rest in bed; especially is this necessary in cases, such as that of J. H—, where hæmorrhage has occurred. As soon as it is safe, quiet moving about in the open air may be allowed, but every form of exercise should for a time be only moderately indulged.

Except in syphilitic cases, drugs are not of great avail. The ordinary remedies are applicable for the relief of sickness, diarrhœa, hæmatemesis, etc., and convalescence is promoted by the administration of iron, quinine, strychnine, arsenic, or the mineral acids, according to indications. It is essential to keep the bowels open daily, and for this purpose nothing is better than small doses of grey powder combined with rhubarb given in the form of a pill at bedtime, and followed next morning by a sufficient dose of Apenta water or other simple saline to insure an efficient result. Drastic purgatives are never desirable. Simple diluents assist the removal of waste products through the kidneys, and should be given as abundantly as the condition of the patient's digestion will permit. For the jaundice, which is such a prominent feature of the unilobular variety of the disease, little more than this can be done. For the ascites, which is a correspondingly important feature of the multilobular type, it is now admitted that early paracentesis offers the most satisfactory plan of management. Varieties of opinion exist as to the best form of trocar to select. In these wards we always use Southey's, and we find them for many reasons both more convenient and more satisfactory than the larger instruments, which are sometimes preferred. When the ascites is of moderate amount and is not rapidly increasing, diuretics may be given a chance. In two recent instances we have attained satisfactory results from the administration of the old Guy's pill, which contains one grain each of digitalis, squill, and blue pill; and in one case a marvellous improvement ensued upon the exhibition of resin of copaiba in twenty-grain doses. Attention has been drawn recently to the value of adrenalin injected intra-abdominally after the withdrawal of the ascitic fluid, but cases in which such treatment is likely to succeed are probably those where the ascites is produced by a chronic peritonitis. Favourable results, from the preventive point of view, are also now sometimes obtained by stitching the omentum to the abdominal wall so as to afford increased facilities for the establishment of a sufficient compensating circulation through the systemic vessels. Other complications and incidental symptoms must be dealt with on general principles as they arise.

July 23rd, 1906.

LÖHNBERG regards stretching of the palate as a notable advance in the treatment of mouth breathing and of chronic occlusion of the tubes. It enlarges a congenitally narrow passage and one that has become occluded from disease. He describes in particular the case of a child of nearly five who was cured of the adenoid expression and was able to breathe through its nose and to hear better after the palate was stretched. — *Journ. A.M.A.*, vol. xlvi, No. 26.

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LECTURES
ON
THE EXAMINATION AND TREATMENT OF EAR DISEASES.

Delivered at St. Bartholomew's Hospital.

By A. E. CUMBERBATCH, M.B., F.R.C.S.,
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Lecture II.

ACUTE CATARRH OF THE TYMPANUM.

Gentlemen, — I want to-day to discuss acute catarrh of the tympanum. It is about the most common of the affections of the ear, and probably there are none of you who have not had it, to some slight extent, though you may not have been aware of it. The commonest cause of all is a cold. Most people when they have a severe cold have a feeling of stuffiness in the ears. They often describe it as feeling as if cotton-wool had been stuffed into the ears, and, as a consequence, that sounds which reach them seem to come from a distance. That is the mildest indication of acute catarrh. In its severest form acute catarrh may produce deafness to a very marked degree. As I have just said, a cold is the commonest cause, but anything which produces naso-pharyngeal catarrh will cause it. Thus you will find it is extremely common after measles, scarlet fever, whooping-cough, and influenza. It occurs after the other fevers also, but those mentioned are the commonest. In other words, 95 per cent. perhaps, of all cases of acute catarrh of the tympanum proceed from the naso-pharynx. In the remaining cases it may be caused by the other things, such as sitting in a draught of cold air in a railway carriage, or driving with a biting east wind blowing into one's ear, or getting cold water into the meatus. These are the chief causes which will produce it through the external passages, but they are not common; nearly all the cases owe their origin to the spread of catarrh through the Eustachian tube. On examining a patient who is suffering from acute catarrh, you find that the meatus is normal in appearance excepting when the attack is a severe one and in that case the postero-superior portion which adjoins the membrana may be pinkish in colour. The meatus all through, excepting that spot, looks quite normal. In slighter cases there may be no alteration in colour at all to the ordinary eye, but the trained eye may notice that the membrana is more cloudy and duller in hue than usual. These are only transient attacks, due to ordinary cold. In a severe case you will notice distinct congestion of the postero-superior meatal wall, and a little later a thin red streak (which is a blood-vessel) running along the posterior part of the handle of the malleus. If the attack is still more severe, a gradual blush spreads downwards till it reaches about the middle and then it begins to creep across the membrana, like a cloud obscuring the sun. Sometimes the redness will involve the whole membrana, so that at last it may be a vivid or dark red colour. If the attack is sufficiently severe for the redness to involve the whole membrana, there is very soon effusion into the deeper layers of the epidermis, and you can see the processus brevis standing out as a white point in the intensely congested membrana; the outline of the handle of the malleus disappears, and the cone of light and the polish of the membrana will also disappear. After a time if there is a great deal of effusion you will notice that the upper segment of the membrana bulges forward. Occasionally in sharpish attacks the effusion lifts up the epidermis from the outer surface of the membrana into irregular patches or little groups of pear-like vesicles, which, however, soon burst. When the inflammation begins to subside the epidermis cracks, and a number of fissures form on the membrana, dark in colour; this darkness is due to the congestion which still exists in the membrana propria being seen through the cracks, while the epidermis itself is quite white. If you test the patient's hearing early in the attack, you will find that in mild cases there is no actual deafness; the watch is heard at the usual distance, or your voice; the sound, however, is not sharply defined, it is muffled. After a time there is real deafness, and you find that the watch is not heard at as great a distance as it should be. If you place a tuning-fork on the vertex of the patient's head—you will remember what I said about examination of the ear in a previous lecture—the watch is heard louder in the inflamed ear. The vibrations are trying to escape through the meatus, but are obstructed in their passage by

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the swollen membrana, and the waves are consequently thrown back, and you get intensification. So that, although the patient hears speech and the tick of a watch worse, he hears the tuning-fork placed on the head better in the affected ear. This shows that the catarrh is confined to the middle ear. Examination of the naso-pharynx will show you that the mucous membrane of the nose and back of the pharynx is congested, and in the case of children you will probably find a certain amount of adenoid tissue. Acute catarrh is much more common in children than in adults, and is frequently limited to one side. Still, both ears may be affected. With regard to symptoms, there probably is little or no fever, except in small children, or unless the case is going to suppurate. The patient may feel seedy, but there may be nothing beyond this in adults to indicate that there is acute inflammation. In a certain percentage of cases there is earache, varying from a dull pain to a sharp stab. Now and then there is tinnitus, generally of a hissing character as if steam were escaping from an engine or a kettle; occasionally the tinnitus is rhythmic—that is to say, the beats of the heart can be heard in the ear. In young children there may be convulsions. In the severest cases after the inflammation has lasted a certain time, one of two things happens: either the inflammation begins to subside, or it becomes intensified, and the fluid, which before was mucous, becomes muco-purulent, and the case becomes one of suppurative catarrh. Although we may suspect, we can never be sure of this till the discharge is seen. With regard to the other symptoms, pain is not common in the ordinary cases, except in children, and they complain of either a dull, aching pain or a sharp stab; but sometimes the pain is very severe, and may be quite severe enough to prevent sleep, and in very young children may even cause an attack of convulsions, giving rise to much anxiety, as, although the convulsions are probably only reflex, they may be due to the spread of the inflammation to the meninges. I told you just now that if there is much effusion there may be bulging of the postero-superior segment. If the pressure of the fluid becomes greater than the membrana, which is weakened by inflammation, can stand, rupture takes place and the fluid escapes into the meatus. If the fluid is non purulent it is either thin, clear, like white of egg, or pale straw colour, and occasionally slightly blood-stained. As soon as the membrana is ruptured and this fluid escapes the pain is speedily relieved, and the edges of the ruptured membrana quickly unite again. Bear this in mind or you will occasionally be puzzled at finding some glairy fluid in the meatus, and yet be unable to detect perforation. This will not often happen, however. Of course, if the fluid is muco-purulent the perforation will not close as long as there is any discharge. If you listen with the diagnostic tube, which you may have to do when you cannot detect the perforation by inspection, while the patient inflates by Valsalva's method, you will probably hear a gurgling sound. If the catarrh does not pass into the suppurative form, you will find that gradually the congestion subsides, the bulging, if present, lessens, and slowly the handle of the malleus reappears, the membrana gradually recovers its polish, but the epidermis may be broken up into irregular patches, and be gradually cast off, and until it is cast off the membrana does not recover its lustre. But even in favourable cases where the patient's hearing is restored the membrana does not always recover its normal appearance. Sometimes the effusion into the deeper layers of the epidermis deposits its salts, and then you find a condition similar to that shown in this picture—chalky deposits in the membrana. The mere presence of a chalky deposit in the membrana is, however, not sufficient to cause deafness. Sometimes the effusion is not entirely reabsorbed, but becomes organised at one or more points, thereby giving an appearance somewhat like that produced in the cornea by a leukoma.

Then, as to the course of acute catarrh. The duration of the inflammation will depend on the severity of the attack and upon the condition of the patient. But in ordinary cases a week or ten days sees the end of it. Still, it may extend to many weeks, especially if the patient is not in very good health or is imprudent. The deafness may vary a good deal. It may be very marked one day and very much better the next. This is due partly to fluctuations in the amount of fluid secreted in the tympanic cavity and partly to the condition of the Eustachian tube—that is to say, whether it is patent or not.

Sometimes after resolution has reached a certain point, for some reason or other, it seems to hang

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fire--that is to say, the patient does not remain very deaf, but, on the other hand, he does not quite recover his hearing; the membrana does not resume its polish, and gradually, sometimes in spite of treatment, the case drifts into one of chronic non-purulent catarrh. That generally occurs in patients who are of a gouty or rheumatic tendency or past the prime of life. You seldom find this happen if the patient is young or in sound health.

Next, as to diagnosis. The only condition with which you can confound acute catarrh of the tympanum is acute myringitis—that is to say, acute inflammation of the membrana.

There is no difficulty, however, in distinguishing between acute myringitis and acute catarrh. In both the membrana is red, and that is the chief point of resemblance between them. In every other respect they differ. For instance, there is practically no deafness at all in myringitis. In the next place, if there is pain it is of a sharp, burning character, not the stabbing and sometimes agonising pain produced by fluid pressure in the tympanum. Pressure on the ear and movements of the jaw in myringitis produce pain, whereas scarcely any pain is caused by such movements in acute catarrh, unless the post-auricular glands happen to be inflamed. Moreover in myringitis you can still see the handle of the malleus, the processus brevis, and perhaps the cone of light, although they may not be so marked; and there is no bulging of the membrana. In very young children acute inflammation of the tympanum is apt to be overlooked. There are no means of finding out whether a baby is deaf or not. It cannot tell you its symptoms. You are called in because it seems ill and fretful. You take the temperature, and find it is raised, and you have to decide what is the matter. One symptom which will often help you is that a small child frequently puts its hand up to the inflamed ear, if it is suffering from earache, and another is that it bores its head down into the pillow, especially towards the affected side. It does not hurt it to do so and it tries to get relief in that way. So if you are called to a young child, and you have thoroughly examined it, and find nothing wrong with its organs, and no sign of any rash to make you suspicious of the presence of any infectious disease, and yet it is ill and crying and obviously in pain, especially if there is a rise in temperature, always examine the ears. If you always do so you will occasionally succeed in detecting what another man has missed. And it is especially important you should do this in children, because it is more likely that an acute catarrh will end in suppuration with them than with adults.

Now as to prognosis. In young people you may safely promise, as soon as it is quite certain that the ear will not suppurate, that the patient will recover with good hearing, because that is the first thing a parent wants to know. He does not know anything about the danger which attends these attacks, or the possibility of suppuration. If it is a very severe attack, you must delay giving a prognosis for a time. But always give your prognosis guardedly in patients who are beyond the prime of life and in those who are actually gouty or rheumatic, or who have any other disease, such as kidney mischief or diabetes. But even in sound people, if there is a history of rheumatism or gout, or if deafness seems to be a family inheritance, always give a guarded prognosis, because those are the cases in which the condition is apt to become chronic.

Now as to treatment. All you can do in mild cases is to insist that the patients shall be kept indoors, and if there is any fever they should be kept in bed. You may give a gentle aperient and apply hot fomentations or a piece of wool made hot and put over the ear. That is often very comforting. This is all that is necessary in a case where there is nothing more marked than deafness. If there is pain, however, you must proceed at once to further measures. The best thing to apply for mild attacks in children is a mixture of chloroform and olive oil in equal parts. Sprinkle a teaspoonful on a large piece of cotton-wool and put it over the ear, not in it, because chloroform put into the ear may set up acute otitis externa. Afterwards wrap the pad round with a handkerchief. If the pain is not relieved in half an hour, put on a second, and if necessary even a third. It will seldom be necessary to apply the mixture more than twice, for one application will generally cut short an ordinary earache in young people. If the earache is more severe you must put on one or more leeches on to the tragus, not on the mastoid as is sometimes done. It is useless leeching the mastoid for inflammation confined to the tympanum. After the leech comes off you may encourage the bleeding to continue for a short

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time. In the severest cases of all the pain can only be removed by relieving the pressure on the membrana, because true earache is the result of pressure on the membrana of the fluid confined in the tympanic cavity. Therefore if the pain is very great, before putting on a leech look at the membrane, and if you find it is distinctly bulging incise it through the bulging portion. If after you have incised it serous fluid escapes wipe this away, and as soon as it ceases oozing through the incision insufflate powdered boracic acid into the meatus. Do not syringe the ear. As the discharge is non-purulent, you, of course, want to keep it so. This incision will probably cut short the severity of the inflammation. If, on the other hand, when you have incised the membrana you find muco-pus escapes, then syringe with some antiseptic lotion, for pus cannot be reabsorbed; it must escape in some direction, and the best direction for it to escape is through the meatus. Therefore, if you find there is muco-pus after incising the membrana, treat the case as I shall tell you when I come to deal with suppuration.

As regards the naso-pharynx, you will examine that and be guided accordingly. If you find in the case of children there are adenoids, then as soon as all inflammation has subsided, and the hearing is fairly restored, remove the growth. If there are no adenoids, but only naso-pharyngeal catarrh, wait until the severity of the attack—supposing the cause to have been cold or influenza—has subsided, and give the patent a simple nasal lotion to douche through the nose. A very good one is borax and bicarbonate of potash, two drachms of each to four ounces of water. A teaspoonful in a wineglassful of warm water can be either sniffed up out of some shallow vessel or it can be used by means of one of these irrigators. This will wash out all the mucus and keep the Eustachian tubes clear. If the patient is not properly instructed how to use a nasal irrigator he will probably find himself swallowing a quantity of the lotion. It will not do him any particular harm, but it is not desirable to drink it. To irrigate successfully the patient should open his mouth and breathe regularly through the mouth. If he does this, the nasal cavity is free from suction, and the lotion will pass up one nostril and flow out through the other.

Next as to the treatment of the deafness. However deaf the patient may be during the acuteness of the attack, do not attempt to do anything for it or even worry your head about it. But as soon as the inflammation begins to subside, if the deafness shows no signs of diminishing, then you must do something for it. Begin to use Politzer's bag, inflating very gently once a day, then once every other day, and so on, gradually diminishing the frequency of it. You will find in most cases that the hearing gradually returns. It may be that the deafness has been simply due to tumefaction of the mucous lining of the Eustachian tube. Supposing the membrana ruptures, or you incise it with this instrument, and pus escapes, then the ear must be syringed night and morning with an antiseptic lotion. You may use biniodide of mercury 1 in 4000, or carbolic acid 1 in 60, or formol in half per cent. Of course there are various other lotions you can use, but those mentioned are among the best. Continue the syringing until the discharge has stopped. Sometimes it will not quite stop, though it may be very slight. In that case change the treatment; do not continue to syringe night and morning, but only once a day, and after each syringing dry out the ear with wool and insufflate boracic acid.

We have been supposing that everything is going well. But everything may not go well. The case you are treating may be one, not of simple but of suppurative catarrh, and the pus may not escape readily through the meatus. It may pass into the attic and through the aditus into the mastoid cells, and then there may be acute inflammation of the mastoid and suppuration there, and the consequences which sometimes follow suppurative mastoiditis. Consideration of that, however, would take us too far to-day. In the next lecture I will tell you about the complications and the dangers which arise.

July 23rd, 1906.

We have received from Messrs. Burroughs Wellcome & Co. a specimen bottle of their "Tabloid" Mistura Alba. Each tabloid contains: Magnesium sulphate 15 grs, magnesium carbonate 2½ grs., oil of peppermint $\frac{1}{32}$ min. "Tabloid" Mistura Alba contains exact amounts of pure, active ingredients, and presents a convenient and reliable means of administering this efficient combination.

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A LECTURE

ON

POTT'S DISEASE IN THE ADULT.

Delivered at Charing Cross Hospital.

By STANLEY BOYD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—Mistakes, like history, repeat themselves; no matter how obvious an error may now appear, it is certain to be repeated, perhaps by one of us. So I have chosen for to-day's lecture a case of Pott's disease with psoas abscess, which latter was regarded as a femoral hernia. I must try to present the case to you as it appeared to the doctor consulted, in order that we may see how the mistake arose and how it might have been avoided; and in endeavouring to do so I avail myself of a note from the doctor, of the patient's statements to myself, and of a certain amount of imagination. The patient is a healthy-looking woman of seventy-six: I should not have been surprised had she stated that her age was sixty-six. For some years past she has kept a sea-side lodging-house and has done a great deal of hard work. A few weeks ago she came up to stay with a married daughter, who seized the opportunity afforded by a cold which her mother contracted to call in her own doctor and to show him a swelling in the right femoral triangle from which the old lady had suffered for about eight months. The only inconvenience complained of was due to pressure of the stays upon the swelling when the patient sat.

You see, then, that the doctor was introduced to a healthy-looking, hard-working old woman, in bed only on account of a cold. On examining the groin, he found a somewhat prominent swelling in the region of the femoral ring which, he was told, began eight months ago as "a very small

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lump" and gradually increased until it became "larger than a hen's egg"—a description which I should imagine would strongly suggest to him a definite circumscribed mass which the patient could take between her finger and thumb. He found that the swelling gave an impulse on coughing; and, although he had some difficulty and was not perhaps perfectly sure about it, he thought he had reduced the swelling. The patient's daughter, standing by, agreed that the swelling had disappeared. What diagnosis could be more natural than that of hernia?

Some pain and increased swelling followed these manipulations and the patient was kept at rest for a few days; then she came up to ascertain the best form of truss to wear.

We noticed nothing unusual in her gait as she walked in. She complained only of a swelling in her right groin, the origin of which she could not really account for—"it might have been due to lifting furniture." She had noticed it by accident, and could not say whether it had ever gone away upon lying down. She laid herself upon the couch quite easily and naturally So far nothing had occurred to cast any suspicion upon the diagnosis which came with her: we were very much in the position of the doctor who made it.

On examining the right groin we found a rounded swelling, of the size and shape of half an orange, occupying the region of the femoral ring. The skin over it was normal. Even to the eye its outline was somewhat indefinite; and, seeing that the patient was thin, Scarpa's space seemed to be unduly full. On comparing the two groins—in accordance with the rule that corresponding parts should always be compared—this fulness became quite certain. Neglect of this rule was probably the first step towards the mistake made. Confining ourselves for the moment to the obvious prominence, palpation showed that it was quite soft and in most directions had no definite margin, though above there was an abrupt, firm edge, which suggested inflammatory infiltration of the deeper tissues here. You could not grasp any swelling, such as the full sac of a hernia: your fingers conveyed the idea of fluid rather than of solid contents. It was not so clearly outlined as a hernial sac containing fluid usually is.

We come next to the second point upon which the diagnosis of hernia was based—viz. impulse.

I did not myself try to obtain an impulse, and there is no mention of such in the notes; but it is possible there was an impulse. Does that in any way pin us to the diagnosis of hernia? Surely not. A true expansile impulse, as distinguished from a shove towards the surface of some non-expanding body, is of the same nature as fluctuation. If you take a sac or bladder full of fluid, lay both your hands on it, and compress it with one hand, you displace a certain amount of fluid under the resting hand, and this hand is consequently lifted up. This is the "fluctuation" which we try to elicit in abscess. In the case of a swelling partly within, partly protruding from the abdomen the abdominal muscles by their contraction replace the compressing fingers. By their contraction they compress the intra-abdominal portion of any such swelling; its contents, if they can, pass out into the extra-abdominal portion and a true expansile impulse is felt. The contents thus displaced may be intestine, omentum, gas, or other intestinal contents in herniæ; pus in abscesses; serous fluid in some hydroceles; blood in large varices—the impulse in all cases is much the same. That being so, it is obvious that the presence of an impulse upon which we lay so much stress in the diagnosis of non-strangulated hernia is not by any means diagnostic of it. I have recently been able to demonstrate to you in the wards the impulse which you sometimes get in one of those large bulging varices of the saphenous or its tributaries, which may occupy so exactly the position of a femoral hernia that they are generally given as one of the things from which a femoral hernia has to be diagnosed. It was smaller than even the prominent part of the swelling which we are considering to-day, but, like it, was very soft and indefinite to the touch, and had no solid contents. It yielded a perfect impulse, because each time the patient coughed the cava and iliac veins were compressed, the flow of blood was dammed back, and in the absence of valves, blood regurgitated from the femoral vein into the varix. Again, this diagram shows an abscess in the sheath of the psoas crossed by the line of Poupart's ligament, and therefore partly inside and partly outside the abdomen. Each time the great abdominal muscles contract they compress its intra-abdominal portion, which is in direct communication with this extra-abdominal, inguinal swelling, and drive some of the contents of the former into the latter, thus

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giving rise to a more or less well-marked impulse in the inguinal swelling. So an impulse in no way binds us to the diagnosis of hernia.

Lastly, the doctor said he was able to reduce the swelling, at all events in part, and the old lady's daughter, who was looking on, confirmed the observation that it was much smaller after than before the attempts at taxis. It is just possible that a hernial protrusion was present and was reduced; or the doctor may have been mistaken, thinking that he had been successful in doing what he wished to do. But let us assume that he was right, and that the prominent swelling was reduced, does that bind us to the diagnosis of hernia? Is a hernia the only reducible swelling that we can get in these parts? Look, again, at this diagram of a psoas abscess, with its divisions above and below Poupart's ligament. Suppose that this abscess cavity is loosely filled, when the patient stands up the lower portion will be full and prominent, but in the recumbent position its prominence may entirely disappear. Pressure upon a diverticulum coming through the saphenous opening would, of course, tend to reduce its contents into the main cavity. But no pressure upon the crural canal, such as would prevent the descent of a hernia, would prevent the re-appearance of a swelling due to a chronic abscess. The above remarks apply equally to the soft, rounded swellings due to varix of the upper end of the internal saphenous or its tributaries: their contents are easily reduced into the main vein, they disappear in lying, and they re-appear on standing in spite of pressure on the crural canal. Almost always, too, there are varices lower in the limb; so from a consideration of these examples only we may conclude that reducibility of tumour in the groin by no means shows that it is a hernia.

We have now considered the grounds upon which the diagnosis of hernia was made; they were, that a swelling was found over the femoral ring which yielded an impulse and was more or less reducible. We have seen that swellings other than hernia may occupy this position and exhibit impulse and reducibility. The diagnosis between these swellings must be made by consideration of other points, and first, the characters of the swelling itself. This was in the present instance soft, ill-defined, incapable of being grasped; its contents were impalpable as free fluid. It is probable that the examiner had no clear idea of a femoral hernia in his mind when he made the faulty diagnosis—the idea of a definite sac, often thickened by a good deal of fat about it, more or less tensely filled with solid masses, such as intestine and omentum, which are usually recognised by the fingers during reduction, if not earlier. Further, and most important, had the two groins been compared it would have been seen that the swelling over the femoral ring was merely a part of a larger swelling which occupied Scarpa's space and the upper part of the thigh.

Hernia being out of the question, what was this swelling? It had no recognisable edge in Scarpa's triangle and the upper adductor region; neither eye nor touch could say exactly where its limits were. The skin of the region, even that over the femoral swelling, was normal; there was no heat or redness, no tenderness or complaint of pain. Fluctuation was evident, though not very distinct, and further examination showed that the fluid lay behind the femoral vessels just below Poupart, and that the swelling was divided by them into an outer and an inner part. Fluctuation was obtained between these two parts, and also from them into the protrusion over the femoral ring. The signs therefore pointed to the presence in the upper part of the thigh of a large collection of fluid which lay outside, behind and internal to the femoral vessels, and which bulged forwards beneath the skin over the femoral opening: the wall of the cavity in which it lay was indicated by a little thickening only at one spot, viz. above the femoral prominence.

What was the nature of the fluid? Almost certainly pus—the pus of a chronic abscess. Blood was out of the question; serous and hydatid cysts would have been well circumscribed and probably subcutaneous. Chronic abscess, on the other hand, is common enough in the groin, and the above description of this swelling is fairly typical. It must, however, be admitted that chronic abscess is not often met with at seventy-six, that no cause for it was suggested by the patient's complaints, or by our observation of her as she walked in and placed herself on the couch, and that there is no family or previous personal history of tubercle. Abscesses which enter the thigh outside the femoral vessels, pass behind them, and then run down in the adductor region, are generally of spinal origin, having come down in the substance of the psoas from some

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focus of tubercular disease. They can usually be detected as they cross the iliac fossa by the presence of fulness beneath the ilio-psoas fascia, and by fluctuation from above to below Poupart's ligament or *vice versa*. So one's hand passes almost instinctively in such a case to investigate the iliac fossa; there was practically no fulness, but I thought there was fluctuation between the swelling in Scarpa's space outside the vessels and an area above the ligament. Certainly it was nothing like so clear as usual in such cases.

Having got thus far, the next thing was obviously to examine the spine, and immediately we looked at it we were struck by a prominence in the lower dorsal region, due to the involvement of one to three bodies. And *now only* did we learn by questioning that pain in the back and a projection of the spine had been present for about eight years, but had been attributed by the patient to rheumatism, and had not, therefore, been mentioned to the doctor or to us. How much more easy the case would have been had she complained of pain in the back and also of a swelling in the groin! And how little would you have appreciated such difficulties as the case presented in practice had I read the hospital notes to you, which *begin* by stating that the patient was admitted "for spinal disease"—probably even less than did the man who thus commenced them!

But the less experienced among you may ask, Do these prominent dorsal spines really mean so much? Do they prove that a patient is suffering from tubercular disease? Not quite. A short, sharp curve like this may result from injury, recent or old, which is easily eliminated: or it may result from gummatous disease of the spine, or from primary or secondary new growth of the vertebræ. Of these, only growth secondary to breast cancer can be described as other than rare. The presence of a malignant growth of any kind in the breast or elsewhere, or a history of the removal of such a growth would make it more than probable that a developing spinal boss was due to a secondary growth. Primary malignant disease of the spinal column is very rare: I do not remember having seen a case. In both primary and secondary malignant disease pain is said to be more severe than in tubercular disease, treatment gives less relief, and of course always fails to arrest the trouble. A positive diagnosis of primary malignant growth will probably be made only when the disease works its way through to the back, and is proved not to be an abscess, or when some secondary growth appears. In connection with this I may direct your attention to a man æt. 40 years, now in Drummond, who for two years past has suffered from what appeared to be ordinary tubercular disease, with a boss in the lower dorsal region. He has suffered singularly little pain for an adult, and, wearing a jacket, has performed light duties in a hatter's establishment practically without interruption. He is thin and pale, but not more so than many a tubercular patient. A few weeks ago he began to have pain in his left groin and thigh, and his doctor, Mr. Maurice Hannay, then found a swelling in the left iliac fossa, and, naturally, supposed it to be an iliac abscess. This swelling was above Poupart, immediately internal to the anterior superior spine, with which it was closely connected. It hardly reached the external iliac vessels. It was smooth, very tense, elastic, and about the size of an orange. It certainly was farther out, more tense, and more circumscribed than most iliac abscesses, but I must confess that I did not seriously consider any other diagnosis. On exploring this swelling through a small muscle-splitting opening, I found it to consist of a soft growth which the microscope has shown to be a round-celled sarcoma. There is still nothing about the "boss" and its neighbourhood to excite a suspicion of malignant disease; so we are in doubt as to whether the iliac growth is secondary to a spinal sarcoma, which is behaving in every way like tubercle, or whether the spinal disease is tubercular and the iliac tumour a primary sarcoma of the hip-bone.

The diagnosis of gummatous osteitis of the spine is to be thought of only when in a case which would otherwise be regarded as tubercular there is a strong history of syphilis, and probably other clear signs of its activity; both are absent in our present case. The occurrence of a "gravitation" abscess, such as was present in the woman whose case we are considering, puts these alternative diagnoses out of court.

On all grounds, therefore, I think we may regard the diagnosis of tubercular disease of the spine with abscess as highly probable. The weakest point in it is the difficulty in demonstrating by the

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fingers the path of the abscess through the loin and iliac fossa into the thigh. But, as we all know, the track through the psoas is sometimes quite narrow and its walls are often very dense, and thus fluctuation is rendered obscure.

The boss of which I have been speaking is median, short, sharp, and angular. It is so important as an indication of disease that we must always be careful not to mistake any other prominence for it. It is due, as you see in these specimens, to the erosion of one to three or more bodies of adjacent vertebræ, the result being more or less sharp flexion of the spine at the point of disease.

Now look at this spine and thorax, also from a young person, which shows another kind of "boss," that of lateral curvature, which is sometimes mistaken for the projection of tubercular disease. There is a marked curve convex to the right in the upper dorsal region, with compensatory curves above and below. As you see, the vertebræ in each of the curves rotate so that their spines project into the concavity of the curve. In the dorsal region the attached ribs move with the vertebræ, and thus in this specimen the angles of the right ribs are carried backwards towards the mid-line and somewhat upwards. This movement is resisted by the attachments of the anterior ends of the ribs, and as a consequence the angles of the right ribs become much more acute and prominent than normal, forming a lump which may be very pronounced, which is never median, and which is always accompanied by the consequential deformities of lateral curvature shown clearly in this lantern slide.

Tubercular disease of the vertebral bodies does not always produce the typical short, sharp curve; sometimes the curve is long, low, and rounded, and then difficulty may arise in distinguishing such curves from similar ones due, say, to rickets in young children or to arthritis deformans in adults, of each of which I show you a photogram. The discovery of an abscess at any one of the points to which spinal abscesses commonly track would, of course, be decisive against either rickets or arthritis deformans.

Lastly, we must remember that tubercular disease of the bodies may produce no deformity for a long time or at all. This is particularly likely to be the case in adults, because in them the bodies of the vertebræ are so large and dense as compared with those of the child, in whom deep erosion is so much more easily affected. Quite commonly in an adult the primary tubercular focus is either actually subperiosteal or so close to the surface of the bone that it soon infects the subperiosteal layer and extends henceforth between the periosteum and the anterior surfaces of more or fewer vertebræ. An abscess, should one form, will lie between the superficially eroded bodies on the one hand and the periosteum, ligaments, and other dense structures in front of the spine on the other. Here is a specimen showing this state of matters in the lower dorsal spine of an adult, and highly suggestive of the possible courses which such an abscess may take: downwards behind the diaphragm and generally under the internal arched ligament, as in our present case; outwards behind the pleura and then either between the transverse processes to issue beneath and at the outer border of the erector-spinæ or, much more rarely, tracking along the intercostal muscles and coming to the surface so far from the spine that its source may not be suspected; or it may burst into the pleura or, should the lung be adherent, into a bronchus and thus be expectorated.

If, then, in our present case we had found no "boss" or spinal deformity, but only the abscess, such as we have described, we should still have had to keep the spine prominently in mind as a possible source of the pus. For, whilst remembering that chronic (tubercular) abscesses are occasionally met with unconnected with bone or joint, their occurrence always suggests to us that bone or joint primarily is at fault. In this instance we should have sought, on the one hand, to show that there were symptoms and signs, other than deformity, of spinal caries and on the other that there was no sign of disease elsewhere which might equally have given rise to the abscess. To take the latter task first: if, as we believe, this abscess lies in the substance of the psoas above Poupart's ligament, the sacro-iliac and hip-joints are the only two at all likely sources other than the spine: and as both joints perform their functions perfectly, we have no reason for suspecting them. So we should next see whether the spine performs its functions—*i. e.* whether it bears ordinary weights easily and painlessly, and moves in all directions freely. We have already ascer-

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tained that for eight years our patient has had more or less constant backache, worse towards night. In her case the pain did not, as it so often does, radiate along the spinal nerves issuing in the neighbourhood of the disease — thus giving rise to complaints of occipital headache, brachial neuralgia, "girdle" sensation, bellyache, sciatica, and so forth. The gait in walking and the way in which some article is picked up from the floor, the way in which the patient sits and lies down or gets up are all carefully watched for evidence of pain or stiffness. Next the patient must stand with the bare back towards a good light and be put through all the movements of which the spine is capable—flexion, extension, lateral flexion, and rotation to right and to left; each movement should be carried to its full normal extent without pain, and the eye and fingers should be unable to detect any rigidity or lack of normal separation and approximation of the spinous processes in any region if the spine is to be regarded as normal. If this examination gives a negative result, and only then, it is desirable to apply further and rougher tests—such as pressing downwards upon the head and shoulders, causing the patient to rise from the lying to the sitting position without using the arms, inviting him to jump from a low height and perhaps even to alight upon the heels. Careful and methodical examination on these lines will in the great majority of cases lead to the recognition of the seat of the disease which has produced the abscess when it is situated in the movable portion of the spine. But it will not always do so because (1) the tubercular focus may be in a part—*e. g.* transverse process—or a portion of a body which is not subject to pressure or friction in movements; or (2) because for some unknown reason pain and the symptoms which result from it occasionally are absent. There is in the museum of the Brompton Hospital for Consumption a preparation showing an abscess surrounding the bodies of the upper nine dorsal vertebræ; at the level of the sixth it opens by a sinus through the right lung into the inferior division of its bronchus. The man was expectorating daily a large quantity of pus, and in the absence of an empyema a spinal abscess was thought of. The spine was examined by various members of the staff—among them my distinguished predecessor Mr. Godlee—but, I am told, without obtaining any positive evidence of spinal disease. The upper dorsal region is naturally the least movable part, and it is here therefore that the greatest difficulty is experienced in detecting impaired mobility; but something more than this is required to explain the complete absence of symptoms and of physical signs in this case in which the spine was almost in two.

The notice of this lecture ran "On a Case of Pott's Disease in an Adult." There are no essential differences between this disease as it affects adults or children, but there are some peculiarities to which attention may be drawn.

Relation to age.—Spinal disease is much commoner in young people than in old, presumably because infection of the old is not so easy. Quite 50 per cent. of the cases of spinal disease occur in the first decade and a decided majority of the remainder in the second decade.

Deformity.—The next point is that adults, as I have said, are particularly liable to anterior disease, and therefore they may escape for a long time, and perhaps entirely, the deformity which is characteristic of the disease. So we must not place too much reliance upon deformity, but be prepared to base a diagnosis upon the symptoms and other signs of spinal disease which we have just considered. Still, absence of deformity leads to a difficulty in the diagnosis of spinal disease in adults which we have not to face so frequently in children.

Suppuration.—Adults are thought to be more liable to suppuration, although in this particular instance suppuration appears to have been resisted for a long time.

Prognosis.—Adults are less liable, so it is said, to throw off tubercular disease when once they are infected; that is to say, the prognosis in spinal disease and in tubercular disease generally is worse as age advances.

Treatment.—Lastly, the size, weight, and obligation to earn a livelihood of an adult add greatly to the difficulties of treatment.

With regard to *prognosis* in tubercular disease of the spine, it depends upon many circumstances, some of which I will mention. I have spoken in general of the effect of age—adult as opposed to childhood—in increasing the gravity of the prognosis. But the age of seventy-six is a very serious factor in any illness, and it is only too probable

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our present patient will not survive the necessarily long period of treatment—if, indeed, she can carry it out. Ability or inability to carry out suitable treatment is another very important factor in prognosis. The more extensive the disease the worse the outlook. Abscess is a grave complication always, and increasingly so as we pass onward from childhood; septic infection of the cavity or sinuses, free suppuration, hectic and albuminoid diseases, make things infinitely worse. Paraplegia is a serious, though not by any means hopeless, addition to a patient's troubles. The general condition of the patient counts for much, one way or the other; and the presence of tubercular disease of the lungs or other important organ necessarily has great weight in making a forecast. Considering her age, our patient is in excellent condition; her spinal disease is *apparently* limited in extent; she has an unopened abscess, but no other tubercular disease that we can discover. Her resisting power is very high, as evidenced by the history of seven years' pain in the spine before any sign of abscess appeared, also by her good general condition in spite of adverse circumstances as regards work and worry. Had she applied for and obtained suitable treatment even six years ago, when she was seventy, I should say that her complete recovery would have been by no means impossible.

I suppose no one has a very large experience of spinal disease in people past mid-life. I can recall only one case, in a woman of fifty-five to sixty. On a voyage to New Zealand in 1898 she fell down the companion ladder and hurt her back. From this she recovered; but later on she had a good deal of rough driving in New Zealand, during which she had much pain in her back. I saw her for continuance of this pain a year or so after the fall, when she had a well-marked "boss" in the lower dosal region. I kept her lying, as much as possible in the country and in the open air, for two years, at the end of which time she began to get about. In the autumn of 1905 she wrote me: "Now it [her spine] is the strongest part of me." If I wished to quote other cases from my own experience, I should have to fall back at once to below forty years of age; but we should not have time to study them. I will, therefore, pass on to the treatment which seems desirable for the patient whose case we are discussing.

The most important point is rest, and without doubt the most perfect rest is given by constant recumbency. Torsion of the spine is to be avoided just as much as flexion, and some patients soon learn instinctively to turn the corresponding shoulder and hip together. When the need for this is not understood or the practice of it is neglected, a suitable corset or other form of splint should be used in addition to the lying position. It must be easily removable for washing. To keep up the limb-muscles massage may be practised.

Next, and almost equally important, are the amplest provision of pure, dry, fresh air, night and day; plenty of sunlight; a simple, easily digested diet, with a large allowance of milk and some cod-liver oil; and attention to every hygienic point.

It is evident that much time and money must be expended to obtain all the advantages indicated in this sketch of treatment even for a child, much more for an adult. Yet it is surprising and very gratifying to see what excellent results may be obtained under conditions which are far from perfect. Two years or more ago I had under my care in Golding Ward a servant girl, æt. 22 years, with sacro-iliac disease and large intra-pelvic abscess. The walls of the joint were chiselled out and the abscess was drained through it. This healed, or all but healed, in hospital, but during the process she developed dorso-lumbar spinal disease. The daughter of a Wiltshire labourer with several other children, we could arrange nothing better for her than that she should return to her father's cottage and there carry out the above treatment. The Charity Organisation Society provided a wheeled stretcher, on to which she is daily lifted from her bed, and on which she lies almost daily in the open. The girl herself understands what is to be done, and with the help of one of the C.O.S. workers has succeeded in drilling her parents into doing it. The reports I have received from her are most satisfactory, and in a very short time now I expect that this patient will be about again. I might quote other cases of serious tubercular disease of large joints with abscess occurring in young adults and doing equally well under similar treatment carried out in one or other of the open spaces of London.

So we shall endeavour to arrange for our present patient to carry out this treatment if and when (as the lawyers say) she leaves hospital. But her abscess required treatment, for it was enlarging somewhat rapidly, and she is, therefore, detained under conditions which are far from ideal. The great thing in the treatment of abscess is to avoid pyogenic infection of the sac, and at seventy-six a long and severe operation is also to be avoided; so I made a small opening outside the femoral vessels, where the coverings of the sac were thick, and evacuated a large quantity of thin pus. I easily found a long, narrow track running up in the psoas sheath towards the diseased spine. The sac was washed out with warm saline, and to the last few ounces a drachm or two of sterile finely-powdered iodoform was added. This was allowed to flow very slowly into and out of the cavity, so that the iodoform, which sinks rapidly, might uniformly coat the walls. The wound was cleaned with saline and carefully closed in layers. A slight rise of temperature, an intermittent pulse, and some depression, passing off in a few days,

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resulted. Some re-accumulation of fluid always occurs: in the most favourable cases it is honey-like, and smells strongly of iodoform; in the least favourable it is pus. But in this case the re-accumulation has been so slight that I have not aspirated or explored its nature in any way. Quite recently, however, ten to fourteen days after the operation, I found the little wound looking full and purplish; there was obviously fluid beneath the thin scar. This was at once opened freely and dressed with wet cyanide gauze: the fluid was pus, but no communication was discovered with the abscess cavity. I sincerely hope that none exists; for if a tubercular sinus forms it is highly probable that it will not heal, but that sooner or later sepsis will be superadded. Several days have elapsed since the re-opening of the scar; the wound appears to be healing, the abscess cavity shows no sign of irritation, and the general condition is undisturbed.

There remain two points for consideration to-day—How long should this treatment be continued? and, How are we to tell when the cure is effected?

The old prescription for spinal caries was "Three years upon your back." If the case did well the time required was probably excessive. In three years I have known a young adult treated as above recover from anterior spinal disease with huge double psoas abscess, and recover sufficiently to ride in a quiet way across country. Without abscess I have known two adults approaching middle age recover after two years' and less than two years' recumbency; so I am inclined to think that in a case which does well two years would be a fair time, after which observations might be made to test recovery; in very favourable cases, perhaps, even eighteen months might suffice for a cure. But it is no easy matter to make sure that the spine is soundly healed. The area of spine affected is, of course, fixed, for it heals by ankylosis. The muscles, however, are not constantly rigid, as there is no longer need for them to be constantly on guard over the tender spine. Pressure may be made vertically downwards upon the head and shoulders and cautiously increased; absence of pain certainly favours, but does not prove, the view that the spine is sound. It is necessary to sit the patient up for a short time at first and then for a longer time, to let him stand, and then walk gradually increasing distances, and finally return to his ordinary life, watching constantly to see whether there is any sign of weakness about the spine as shown by a desire to spare it and to take weight off it. Much nice discrimination must be shown in deciding this point, for general weakness and fatigue and all sorts of aches and pains are certain to be complained of when a patient leaves a couch after two years upon it.

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SOME REMARKS ON THE TREATMENT OF ACCIDENTAL HÆMORRHAGE AND ECLAMPSIA.*

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GENTLEMEN,—When your Secretary (Dr. Osborn) so kindly asked me to come here to-night he threw out a hint that I should choose for discussion something that was not too rare, and, further, that it would be as well if I would introduce two subjects. He has been good enough to agree to those I have chosen—namely accidental hæmorrhage, and eclampsia. Though not common in the practice of any one of us, neither of these complications of pregnancy can be said to be rare. Occurring suddenly and requiring immediate treatment, it is well to have clearly before us the line of treatment we shall adopt in each circumstance.

I will first of all consider the subject of *accidental hæmorrhage*. Bleeding due to placenta prævia I have not touched upon, because the treatment of this complication has not materially altered since Dr. Braxton Hicks introduced the method of dealing with it by version.

The treatment of accidental hæmorrhage has undergone of late years a radical change in the hands of some of us. The classical method of dealing with these cases was by rupturing the membranes, and this still occupies the place of honour in most of the text-books. No doubt in many cases this effects its purpose, and by causing a cessation of bleeding does away with the immediate risk to life. That it is not regarded as a uniformly successful measure may be seen by the frequency with which *accouchement forcé* is recommended and has been adopted in actual practice.

Let us consider first of all a case in which the hæmorrhage has started before the onset of labour. We have ruptured the membranes and the uterus has contracted down, with the result that bleeding ceases. We are now faced with the probability of a tedious labour. The cervix is not dilated, the

* Delivered before the Dover Division of the South-Eastern Branch of the British Medical Association.

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waters have drained away, and the preceding loss of blood will not conduce to active uterine contractions; in fact, we have every prospect of a long attendance on a case which we dare not leave. And, further, supposing that hæmorrhage recurs later we are faced with the prospect of forcible dilatation of the cervix and the extraction of the child.

But there is another point to bear in mind and a more important one still. In some cases after rupture of the membranes the uterus does not contract down but remains inert and flabby, and we are then faced with a state of affairs that is very much worse than existed before the liquor amnii escaped. For now we have a flaccid uterus into which blood can escape freely, such pressure as the bag of membranes previously exerted being now lost. Here, then, we are faced with a desperate condition, the only chance for the patient being an *accouchement forcé*.

Can we decide beforehand the cases in which the uterus will contract down and those in which it will not do so? I do not think we can decide this point with any degree of certainty; but if we elect to rupture the membranes, I would say, do so only in those cases in which active uterine contractions can be made out—that is, in those cases in which labour is in progress. As for *accouchement forcé*, namely the forcible and rapid dilatation of the cervix and prompt delivery of the child, this is a procedure which in a private house and without adequate assistance and instruments, is not one which one could anticipate with anything but the greatest anxiety.

Now, have we any means of avoiding these difficulties? I think we have. Since I adopted a method of treatment that came over to us from Dublin a very few years back I have met with no difficulty and have had very little anxiety in connection with the mana gementof these cases.

This was a return to an older method of the treatment of uterine hæmorrhage by plugging the vagina. It was employed largely for the treatment of placenta prævia before Dr. Braxton Hicks introduced bipolar version for that purpose, and was given up as a routine treatment on account of the septic complications that attended its use. But asepsis in those days was not understood as we understand it now and the results are not to be wondered at.

But the old dread of the vaginal plug has remained till to-day, and there are those who still denounce it, their dislike to its use being based, I have no doubt, on the experience of the pre-antiseptic days. I have seen no ill results from its employment, and if the plugging is carried out with a due sense of asepsis it need not carry with it any risks.

It is interesting, again, to recall the fact that till quite recently the mention of vaginal plugging for accidental hæmorrhage would alone have been sufficient to bring about our rejection at an examination on this subject. It was supposed, and, in fact, stated positively, that this procedure would convert an open into a concealed accidental hæmorrhage, the most dreaded in all probability of all complications. That this was pure supposition is shown by the fact that in no instance recorded has this taken place.

I have employed this method for the last three or four years, and in that time I have not had a case which has given me any anxiety.

The first case I treated in this way was a very stout woman approaching middle age, who had bled very freely and in whom I could detect no uterine contractions. Here was, I thought, as unfavourable a case to puncture the membranes as one could choose. She was pallid from loss of blood, and it appeared not improbable that the uterus might fail to contract. I consequently plugged the vagina firmly, put on a binder, and gave her some opium, as she complained greatly of the discomfort. In two hours' time she expelled this plug, which was quickly followed by the child and placenta. This result was to me a surprise, more especially as regards the rapid onset of labour. There was no further bleeding or cause for anxiety. Within the next two years I repeated the same line of treatment on this patient for a second attack of accidental hæmorrhage. This is typical of other cases, though the children have not always been born as speedily. I should say, speaking roughly, that labour starts, as a rule, within six hours of plugging.

Now, what are the advantages of this method of treatment?

To begin with, the plug and binder, properly applied, are most efficacious in arresting the bleeding.

Secondly, it makes little difference whether the

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uterus is contracting strongly or not, and consequently we have not the risk that attends rupture of the membranes of bleeding persisting. Further, it allows of a period of rest during which the uterine muscle recuperates its powers. This is of great importance. Lastly, it acts as a strong stimulus to labour and leads to quick dilatation of the cervix.

Thus it serves most efficaciously the two main indications in treatment—the arrest of hæmorrhage and the induction of labour. In fact, it is a method that is easy of application, minimises the risk of subsequent bleeding, and obviates the tediousness of labour with the risks to mother and child that attend premature rupture of the membranes.

But in its application there are certain points to attend to. In the first place no detail in asepsis should be overlooked. A hot antiseptic douche should first be given to wash away blood-clot, the heat probably helping to bring about uterine contractions and relaxation of the cervix. Next the material for plugging should be some antiseptic gauze, iodoform for instance, or some material, such as wool or gauze or linen, that has been rendered aseptic. This can be effected readily by boiling, the material used being wrung out of an antiseptic solution before use. The plugging itself is best carried out with the help of a Sims' speculum. It is worth noting that the handle of a tablespoon makes a very good substitute for the purpose in an emergency, and was, I believe, Sims' first idea for his speculum. Either strips of gauze or pledgets of wool are taken and packed firmly around the cervix in the fornices, the vagina being subsequently firmly filled. A tight abdominal binder is next applied and a perineal band to support the vaginal plug, though I have not always used this latter. If there is much pain give opium. It will be an advantage to the patient if she can get a little sleep after the bleeding, and she should be encouraged to have some fluid nourishment. I do not think the plugs should be left in longer than twelve hours if labour has not commenced by that time. In the majority of cases, however, labour starts before that time has elapsed. If it has not done so, remove the packing, give a vaginal douche, and replace the plug. All measures should be adopted to minimise the risk of post-partum hæmorrhage, and these I need not particularise here. I may add that I have not had any septic trouble from the use of the plug in these cases. So far as rupture of the membranes is concerned, I would say here that it is a good and proper method to employ when the patient is in strong labour at the time of the bleeding. It stimulates uterine contractions and is supposed to prevent the further detachment of the placenta that the advance of the entire bag of membranes would be likely to promote.

I will now pass on to the subject of *eclampsia*. Here, I think, we meet with greater difficulty than in accidental hæmorrhage; for in the latter at all events a definite line of treatment was laid down in the past for our guidance, whereas in the case of eclampsia we may say that there are as many opinions as there are men who preach them.

If there has been one guiding principle in the past it has been this: that eclampsia being connected with pregnancy, for its relief the pregnancy should be forthwith ended; and this rule still holds with many, more especially abroad, and leads to every variety of forced labour from manual dilatation of the cervix to Cæsarean section. That eclampsia is specially connected with pregnancy there is no doubt; that forcible delivery in all cases is the best method to adopt is by no means so certain.

Roughly, there are three groups into which fits of an epileptic character fall:

(1) Cases in which epileptic fits occur without albuminuria and without any evidence of disease in other organs. These cases are indistinguishable from ordinary cases of epilepsy.

(2) Cases in which, with the fits, there is marked evidence of a kidney lesion as shown by a diminution in the amount of urine, albuminuria, headache, nausea, and varying degrees of anasarca. On the whole dropsy is not a very marked feature, and we may say that the more dropsy there is the less liability there is to fits.

(3) There is a third group in which the symptoms appear to be referable to a derangement of the liver—a hepatic type. In these cases the urine shows slight albuminuria only or none at all, and jaundice is a noticeable feature. These cases are rare, and are interesting mainly as showing that the condition underlying eclampsia is a widespread one and affects organs other than the kidneys.

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I cannot go at any great length into the pathology of eclampsia; but as the treatment must be based on a knowledge of the essential factors underlying its causation, I must refer to these, and shall do so as shortly as possible.

The chief factors concerned in the production of fits are as follows:

(1) First of all the increased irritability of the nerve-centres in pregnancy. This is shown in many ways. I need only mention such conditions as the irritability of pregnant women, chorea, hysteria, and lastly, mania. To this no doubt must be attributed in the main the first clinical group I mentioned, in which epileptic fits occur, perhaps, for the first time in a patient's history—fits unassociated with any evidence of lesion of other organs and indistinguishable from ordinary epilepsy.

(2) Secondly, we have as a factor a condition of toxæmia. In true eclampsia as distinguished from epilepsy this is the real underlying factor and the one on which most stress must be laid. The whole course of the disease and its clinical picture is one of toxæmia, commencing as it does with headache and nausea and ending in convulsions and coma.

The association of fits with albumen in the urine has long been known, and they were thought to depend on a primary failure of the renal function. This failure was attributed to pressure on the ureters or the renal vessels or to reflex disturbances of the kidney. There seems to be little doubt however, that in the majority of cases, at all events, the kidney lesion is not primary but is the result of toxins produced elsewhere, which in the course of circulation or elimination affect the renal parenchyma, causing changes of an inflammatory and degenerative nature similar to those produced by other toxic states such as scarlet fever.

Although the renal changes are not to be regarded as primary, but rather as one of the results of a toxæmia, the effects of which are manifested in other organs, they are of great importance, for they lead to failure in the elimination of the toxins and further accumulation of these in the system.

That the kidneys are not the organs essentially at fault is shown by those cases, comparatively uncommon, in which there is no serious interference with their working.

Another organ to which much attention has been drawn of late is the liver. Here hæmorrhages and degenerative changes varying in degree are found, these sometimes being so extensive as to resemble the changes found in acute yellow atrophy. The discovery of the changes naturally led to the view that the liver was primarily at fault and that the hepatic function being deranged, products of metabolism ordinarily dealt with by that organ escaped unchanged. It seems more probable that these changes in the liver, as is the case with those in the kidney, are to be regarded rather as incidents of the disease than as the primary cause. In fact, degenerative changes may be found widely distributed and they are such as we associate with general infective and toxæmic states. Where the toxin is produced we have not at present such evidence as would enable us to state with certainty. It may be the result of some failure of metabolism in the mother, or it may be elaborated in the uterus by the fœtus or placenta. The clinical evidence put forward in favour of the latter is the actual association with pregnancy, the amelioration of symptoms that has been supposed to follow the death of the fœtus, and the beneficial effects that are largely held to follow the emptying of the uterus. Against this view we have the facts that fits may cease, and often do cease, whilst the fœtus is still in the uterus and alive, and that fits may not supervene till after labour is completed.

From the experimental side evidence has of late been brought forward to show that the placenta in eclampsia contains highly toxic substances. Future work will, no doubt, clear up the question of origin of the toxins. It is possible that there may be more sources of production than one. All that we can say with safety at the present time is that toxic substances are produced somewhere. These circulating in the system produce degenerative changes in the viscera. Owing to failure of elimination by the kidneys, or to changes in the liver, that prevent its dealing successfully with toxic material brought to it, these toxins accumulate in the system and, acting on the nervous centres, produce convulsions.

We ought not to forget with regard to this question of elimination the importance of the skin and bowels. It may be that some failure in these may be the determining cause of a fit. I saw some time ago a patient with much ascites

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and about full time. She had had an attack of diarrhœa, a providential event, no doubt, for her. It had been checked, unfortunately, by means of drugs, and its cessation was followed immediately by the convulsions of eclampsia. Here, in all probability, the free action of the bowels was just sufficient to keep the toxins in the system below the amount necessary to produce fits. As soon as it was checked, these accumulated, the kidney elimination being very defective, and eclampsia supervened.

(3) The third factor in causation is the presence of the fœtus and placenta *in utero*. Its importance depends on two points: (*a*) whether or not toxins are produced by the fœtus or placenta; (*b*) whether the pregnant uterus is the starting-point of impulses which, acting on irritable nerve-centres, cause convulsions.

The former I have already referred to. The importance of the latter is very difficult to estimate. No doubt convulsions occasionally occur in connection with obstructed labour, in young or old primiparæ, or when labour is very painful. The tendency, no doubt, at the present time, is to lay less stress on this as a possible factor. Its importance is probably small as compared with other conditions mentioned.

I will now pass on to the subject of treatment. You will see from what I have said that I consider the most important factor to be the toxæmia present. The tissues are loaded with a poison that is not being eliminated. Most often the kidneys are working very imperfectly, a small quantity of urine being excreted loaded with albumen.

It seems reasonable therefore that our first endeavour should be directed to getting rid of the poisonous materials as quickly as possible through the skin and bowels. To this end we at once administer a purgative. I must admit here that the result is not always as satisfactory or as rapid as one would wish. If the patient is sensible she can take five grains of calomel, and to this may be usefully added a drachm of Pulv. jalapæ co. If she is insensible two minims of croton oil mixed with a little butter is generally employed, and is placed on the back of the tongue. The patient often froths this up and spits it out and I think a better plan is one recommended by Mr. Jellett, to mix it with some olive oil and give it by means of an œsophageal tube. This should be followed by an enema. The patient should be wrapped up warmly, and to promote the action of the skin a hot bath should be given, or better a hot air bath or hot pack. At the same time a careful watch should be kept on the temperature, for this rises when fits are numerous and hyperpyrexia may supervene. If the patient is sensible between the fits encourage her to drink copious draughts of fluid. There is another method of introducing fluid, that has been introduced by Dr. Jardine, namely infusion of saline fluid. This can be passed direct into a vein, but I prefer to pass it into the cellular tissue of the axilla, as being much easier to carry out and generally quite efficacious. A pint can in this way be quickly introduced into each axilla and can be followed up a little later by fresh infusions if required.

At the same time as we are preparing to carry out the above treatment we must consider measures for controlling the fits. The three chief agents we employ for this purpose are chloral, or chloral combined with bromide of potassium, chloroform and morphia. Of these I prefer morphia. Half a grain should be given, followed by quarter-grain injections if the fits continue. As much as three grains may be given apparently in the twenty-four hours, though I have never given as much as this. The patients bear it well, and it has not the depressant effects that chloroform and chloral have. Chloroform has its uses, however, and should be employed when any operative procedures have to be carried out.

We have now given morphia to control the fits and have done what we can to promote the free action of the skin and bowels. And we come now to the question of emptying the uterus. If the patient is in labour, by all means terminate it as soon as possible. As soon as the os is sufficiently dilated put on forceps and deliver.

But if the patient is not yet in labour what are we to do? At the present time opinion is divided on the question of immediate emptying of the uterus. On the one hand are those who follow the line of treatment described above, leaving the labour, so far as possible, to occur spontaneously. For some time now I have seen most excellent results follow this line of treatment. On the other hand are those who resort at once to forcible delivery of the child. There is no doubt that the introduction of Bossi's dilators has facilitated this mode of delivery, though some operators who have obtained good results prefer vaginal Cæsarean section. I have been looking up some of the most recent statistics to see how they help us.

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Till quite recently the mortality seemed to be somewhere between 20 and 30 per cent. whatever mode of treatment was adopted. Some figures given us latterly show better results, and I will first take those collected by Dr. De la Harpe at the Rotunda Hospital. The treatment there adopted for some years is that described above, and the mortality in 71 cases was 16·9 per cent. This undoubtedly is a good result and apparently includes all cases admitted. The importance of early treatment is emphasised, the mortality being mainly due to patients who were admitted late in a moribund condition. With early treatment on these lines there seems every hope that the above death-rate might be materially lessened.

When we come to the results of treatment by immediate delivery abroad, it is, to say the least, startling to find that in Bumm's clinic in Berlin the mortality has been reduced to 1·8 per cent.

The two sets of figures can scarcely be compared. For in the latter, cases only that were seen shortly after the commencement of the attack were included. Thus all cases occurring after delivery, those seen for the first time six to twelve hours after the commencement of the attack, and cases dying of aspiration pneumonia were left out. From the number of cases dealt with, eclampsia would appear to be a more common complication abroad than it is here. It is possible that slight cases occur here and are dealt with successfully at home that abroad would be treated in hospital. At all events, we do not know how the average severity of eclampsia abroad compares with the disease in this country. Until we have some figures in regard to the former method of treatment drawn up as exclusively as those given for immediate delivery it is impossible to compare the two. At present I am satisfied with the treatment I have described above and I am hopeful as to future results. I do not wish to be dogmatic and it is possible that the logic of facts may later emphasise the necessity for immediate delivery. I am to-day concerned mainly with methods that can be easily carried out in private practice. *Accouchement forcé* is one thing in the hands of expert operators who have had experience of very many cases—and no doubt to obtain such results as those quoted above considerable manipulative skill must have been displayed. It is another thing when attempted very rarely by those not accustomed to such operations. For the present I would advise the simpler measures I have described. Take the case promptly in hand, give morphia, attend to the skin and bowels, give saline infusions, or failing these, copious rectal injections; and you will have done what lies within your power to do easily, and what, moreover, is justified by such knowledge as we possess of this complication at the present time.

July 30th, 1906.

ETHYL CHLORIDE—ITS USES AND ABUSES.*

By J. H. CHALDECOTT, L.R.C.P.,
Anæsthetist Italian Hospital and Royal Ear Hospital.

MR. PRESIDENT AND GENTLEMEN,—The introduction and rapidly increasing adoption of ethyl chloride as a general anæsthetic in this country constitutes the most important innovation which has occurred in the domain of anæsthesia during recent times—a fact made the more remarkable when we consider that for some years previously it had been used in different parts of the Continent without attracting any particular notice here. Another curious point in this connection is, that even after there had been several papers published on the subject the profession made no substantial move in the matter until the attractions of the drug were systematically advertised by chemists and others who were interested in the sale of somnoform.

In 1901 a paper upon "Ethyl Chloride" was published by Dr. MacCardie, and in May, 1902, a paper upon "Somnoform" was read by Drs. Rolland and Field Robinson before the British Dental Congress at Shrewsbury. Although these papers induced a few to give a trial to ethyl chloride, they do not seem to have attracted very general attention, as in June, 1902, a paper was read by Dr. Mortimer on "One Thousand Administrations of Anæsthetics" before a large meeting of the West London Medico-Chirurgical Society, and during the discussion which followed I inquired whether he or anyone in the room had tried ethyl chloride in general anæsthesia, the answer being in the negative. I had myself at that time used it in conjunction with my colleague, Mr. George, at the Central London Throat Hospital, but we had obtained very uncertain results, owing, as it afterwards transpired, to the unsuitability of the inhaler we had employed, so that then I was in a position to do no more than hint that the drug might not improbably be found useful in certain cases.

In April, 1903, a second paper was published by Dr. MacCardie, which contained the valuable point of information that he was in the habit of

* Delivered before the Society of Anæsthetists.

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administering it from a closed inhaler, an example which many of us lost no time in following, with immediately improved results. On the same day upon which this article appeared in the 'Lancet' a paper on "The Use of Somnoform" was read by Mr. Foster Cross, who was, however, by no means enthusiastic on the subject, concluding his paper by stating that the experience he had had of the anæsthetic did not lead him to advocate its use. In the course of the discussion which followed Mr. Cross's paper, only three speakers could claim much experience of either somnoform or ethyl chloride, but all of these had favourable results to report.

In October, 1903, I read a paper on "Ethyl Chloride" to the Society for the Study of Disease of Children, in which I strongly advocated its more general adoption, but it is probable that at that time and until the following year *somnoform* was being used much more largely than ethyl chloride, owing, doubtless, to the fact that it had been so largely advertised. Since the beginning of 1904, however, ethyl chloride has rapidly increased in popularity, and is at the present time very much more largely used than somnoform. My subsequent remarks must be taken as applying to pure ethyl chloride, and not to somnoform.

With reference to the uses of ethyl chloride I wrote two years ago that, having then administered it 500 times, I had arrived at the following conclusions:

(1) It is the best anæsthetic known for single-dose cases, viz. those in which anæsthesia is induced, the mask removed, and the operation performed without any further administration. (2) It is certainly worthy of an extensive trial in all minor operations. (3) It is a safe and rapid means of inducing anæsthesia as a preliminary to chloroform or ether narcosis. (4) Although its effects are transient, it is doubtless a powerful anæsthetic, and should be carefully administered and the patient properly prepared.

Since that time I have multiplied very largely my experience of the drug without materially altering my opinion on any of these points, which were then intended to apply only to children, but are equally applicable to the case of adults.

With reference to the first of my conclusions, viz. that ethyl chloride is the best single-dose anæsthetic, I still adhere to this opinion in the case of children. For adults of an ordinary type nitrous oxide still stands, as Dr. MacCardie has expressed it, in a class by itself, and for all operations where gas will fulfil every requirement I think we ought to employ it, but of course even in adults there are very many short operations upon the mouth and nose, and some upon the eye, which cannot be satisfactorily performed under a non-continuous gas anæsthesia, and it is here that ethyl chloride, either by itself or combined with gas, has proved such a valuable addition to the stock-in-trade of the anæsthetist.

In one department alone of minor surgery I believe ethyl chloride is destined to save many lives. I allude to operations for enlarged tonsils and adenoids. The chloroform mortality in these operations is exceptionally high. Between 1890 and 1900 over a hundred deaths were recorded in Great Britain as the result of chloroform administered for adenoid operations, chloroform appearing to present peculiar danger to patients of the "adenoid" type.

On the other hand, nitrous oxide and ether are by no means ideal anæsthetics for these operations, on account of the cyanosis and secretion of mucus which they cause, but in ethyl chloride we have, I think, an anæsthetic which is as nearly as possible perfect for this class of surgical work, providing as it does a longer anæsthesia than gas or gas and ether, and one of a better and quieter type.

For "single-dose" cases in adults, except in the case of big muscular men, I prefer the nitrous oxide and ethyl chloride mixture, my reason for this preference being that equally good results can thus be obtained with smaller doses of the ethyl chloride than when the drug is administered by itself, the after-effects being thereby minimised. I regard this mixture as an ideal anæsthetic for cases requiring an available anæsthesia of from forty seconds to two minutes.

With reference to the second of my conclusions that the drug was worthy of a trial in all minor operations it must, I think, be admitted that it has now had a fairly extensive trial, and in some hands at any rate has given very favourable results. Personally, however, I do not see any advantage in continuing the administration of ethyl chloride after the first few minutes of anæsthesia: it is, I think, more difficult to keep up a level type of

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narcosis with ethyl chloride than with either ether or chloroform, the extreme volatility of the drug making it difficult and irksome to administer methodically, and I do not know of any counterbalancing advantage, as I am inclined to think that the after-effects of continuous ethyl chloride narcosis are practically identical with those of chloroform.

My third conclusion was that it is a safe and rapid means of inducing anæsthesia as a preliminary to chloroform and ether, but on this point I do not now feel quite so confident. Of the convenience and rapidity of this method there can be no doubt, and, as a consequence, the ethyl-chloride-ether sequence has become so popular that at some of the hospitals the gas-ether sequence has been practically abandoned. I feel, however, that in the light of recent events we ought to consider very seriously whether the ethyl-chloride-ether sequence is not attended by danger to life which does not exist when gas and ether are used. At least two of the fatalities under ethyl chloride have happened during induction by this method, and there have probably been many narrow escapes; I have myself seen one in which artificial respiration was necessary.

Although it is quite possible that these deaths were due to over-dosage, and were, therefore, preventable accidents, still, the fact remains that we are substituting a method in which a slight mistake may lead to a fatal result for one which is practically safe.

I do not, therefore, think that the ethyl-chloride-ether should be substituted for the gas-ether sequence except in cases in which for some reason connected with the type of patient or the nature of the operation nitrous oxide is contra-indicated.

In certain cases in which rapidity of induction is essential the ethyl-ether or ethyl-chloride sequence has distinct advantages. I recently had on four occasions to anæsthetise a man who was suffering from acute maniacal symptoms due to a cerebral abscess. Gas and ether were contra-indicated, and, owing to his struggles, it would have been by no means an easy task to get him under chloroform with safety. I decided to give him ethyl chloride, and on each occasion his struggles ceased after two or three inspirations, and he passed rapidly and quietly into a state of deep anæsthesia.

My fourth conclusion, that ethyl chloride is a powerful anæsthetic and should be carefully administered and the patients properly prepared, is fully recognised now, and needs no further argument.

Now, what are the objections which have been raised to ethyl chloride? The only ones I have seen or heard mentioned are:

(1) Uncertainty of action.
(2) Rigidity.
(3) After-effects.
(4) Danger to life.

The first of these objections—*i. e.* uncertainty of action—was raised by Dr. Hewitt, who a year or so ago published an account of a series of cases in which he had tried various mixtures of ethyl chloride and air. These experiments were carried out with the most admirable attention to detail, and from an experimental point of view were interesting; but from the point of view of determining the clinical value of ethyl chloride they were, in my opinion, most misleading. For at least twelve months before the publication of these experiments it had been recognised that in order to obtain the best results with the drug it was essential for it to be administered with a very limited supply of air; none, however, of Dr. Hewitt's cases had the drug administered to them in the ordinary way, 3000 c.c. of air being the minimum allowance, so that it is not surprising that in no single case did he achieve a really satisfactory result, forty-two seconds being his average available anæsthesia. As one would expect, all his cases showed a longer induction period and a shorter available anæsthesia than when the drug is given from a smaller closed inhaler, such as an Ormsby. Unfortunately, on the strength of these cases Dr. Hewitt published as one of his conclusions that "Ethyl chloride is somewhat uncertain in its action, sometimes answering every possible requirement and fully satisfying the patient and his medical attendants, at other times almost failing to produce anæsthesia." Now, this conclusion is identical with the opinion which was formed of it by those of us who some years ago were trying to administer it with unsuitable inhalers, and the cause of Dr. Hewitt's failure was also identical with ours—viz. too much air. I am certain that if Dr. Hewitt had taken another

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series of cases, and, having abandoned his specially devised apparatus, had employed any small closed inhaler such as an Ormsby or an adapted Clover, he would never have published as his opinion that ethyl chloride is uncertain in its action. Whatever may be the faults of ethyl chloride, uncertainty of action is not one of them. We have no other anæsthetic agent with which we can be equally certain of inducing anæsthesia within so short a space of time, and I will go so far as to say that failure to produce deep anæsthesia within sixty seconds, even in the case of a muscular alcoholic, means a fault of some kind in the administration.

I have heard it stated that one of the objectionable features of ethyl chloride is that it produces *rigidity* more than other anæsthetics. I am bound to say that I cannot understand anyone who has had much experience of the drug holding this opinion. It may be assumed that there is a stage of rigidity during the administration of all anæsthetics, but it appears to me that one of the most striking characteristics of ethyl chloride is its power of practically abolishing this stage by the rapidity with which it induces complete anæsthesia—flaccidity is reached so quickly that no stage of rigidity is noticed. On this point I am anxious to hear the testimony of others who are in the habit of giving the drug to large numbers of cases, whether as a matter of fact they have not found that absence of rigidity is one of the most striking features in its administration.

The objection which is based upon "after-effects" is an important one. The most commonly heard of "after-effects" are vomiting, headache, post-anæsthetic faintness, and a general feeling of malaise.

In estimating the significance and frequency of these symptoms it must be borne in mind that during the first two or three years following the introduction of ethyl chloride and somnoform into this country a very faulty estimate of their uses and properties was formed, trading firms who were interested in their sale having issued pamphlets broadcast to members of the medical and dental professions which were exceedingly misleading. The statement was frequently made that ethyl chloride, while superior to nitrous oxide in producing a longer and deeper anæsthesia, was at the same time equally innocent of after-effects, and might with impunity be administered to patients who had undergone no special preparation. As a result the drug was very largely abused and many cases of post-anæsthetic vomiting and other disturbances were reported which would, with ordinary discretion, have never occurred.

On the other hand, if we disregard cases in which the stomach is full and in which blood has been swallowed, I believe the after-effects of single doses of ethyl chloride are very slight and very transient. I have on two occasions in my own practice met with vomiting lasting several hours, but in one case the patient against advice had a good lunch about an hour before operation, and in the second case the ethyl chloride was continued for about ten minutes. On a subsequent occasion I gave this patient chloroform, and she again suffered very badly from vomiting after a comparatively short administration.

However, nausea and perhaps retching lasting a few minutes are not uncommon, and it is in this particular respect that the gas and ethyl chloride mixture shows its superiority, it being in my experience quite exceptional for a properly prepared patient to vomit after taking this mixture unless blood has been swallowed during the operation.

It is possible that this comparative absence of after-effects when the mixture has been given may be due to greater accuracy of dosage, the exact amount of ethyl chloride being always measured off into the little receiving tube at the end of the bag; and it occurs to me that if this were always done even when the drug is given "neat," it is probable that we should hear far less of after-effects, and possibly avoid fatalities altogether. When the drug is sprayed directly into the bag it is not easy to regulate the dose with exact precision and in consequence too much may easily be given.

The last but by no means the least important objection which we have to consider is that which arises out of the fatalities which have occurred under ethyl chloride. Although the number of fatal cases represents an infinitesimal precentage of actual administrations, yet it is sufficient to show us that the drug offers an appreciable mortality, and to give us food for reflection. For instance, ought we unless for special reasons to discard the gas-ether sequence so frequently? And, if ethyl chloride has an appreciable mortality, ought we not to discourage as far as we can its haphazard administration by those dentists who are not qualified medical men, and who have had no anæsthetic training?

July 30th, 1906.

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A CLINICAL LECTURE

ON

CEREBELLAR DISEASE.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, W.C.

By J. S. RISIEN RUSSELL, M.D., F.R.C.P.,
Physician to Out-Patients at the Hospital, and Physician to University College Hospital.

LADIES AND GENTLEMEN,—The subject I propose to discuss with you to-day is cerebellar disease.

The cerebellum is, of course, liable to diseases which affect other parts of the nervous system. There may be vascular lesions of the cerebellum as of the brain; a vessel may become occluded or it may rupture, and thus there may result softening or extravasation of blood into the substance of the organ. These, however, are rare conditions. When they occur they give rise to clinical pictures which are highly characteristic, and from which we may learn a very great deal in regard to the symptoms that result when the cerebellum or its peduncles are damaged by disease—cases of thrombosis are especially valuable, for in vascular occlusion we have a lesion which causes a defect in the organ which is not complicated by the effects of increase of intra-cranial pressure. Cases of this kind, however, are rare, and, therefore, we have not many of them to help us in the study of the symptomatology of diseases of the cerebellum. Those that have been recorded help to teach us what kind of clinical picture we should expect when there is cerebellar insufficiency. The cerebellum may also be the seat of gross lesions, such as tumours and abscesses, of which we shall have to speak more in detail presently. The organ may also undergo atrophic changes, as, for instance, in the disease known as hereditary cerebellar ataxy, in which, in conjunction with certain sclerotic changes in the spinal cord, there are also atrophic changes in the cerebellum itself, a state of things that leads to a

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definite clinical picture by which this malady can be recognised. We may also find the cerebellum in an atrophic condition owing to developmental defects which affect it alone or in conjunction with the brain, and which may give rise to a characteristic clinical picture, although it is surprising how little there may be that can be said to be characteristic in some of these cases. Finally, we must recognise that the cerebellum may be the seat of sclerosis. It may be so affected as part of the general disease disseminate sclerosis, in which, as you know, patches of sclerosis may occur in any part of the nervous system, and a certain colouring may be given to the clinical picture of that malady owing to changes of the kind occurring in the cerebellum or in one of its peduncles. Thus, in a disease like disseminate sclerosis we may find certain manifestations which enable us to say that in all probability there are morbid changes in the cerebellum or its peduncle; indeed, I have seen cases where cerebellar tumour has been diagnosed, and the advisability of an operation considered, when in reality the case was one of disseminate sclerosis. I mention this to emphasise how closely the clinical picture may resemble one of cerebellar disease, when in reality it is due to disseminate sclerosis in which the cerebellar manifestations are so prominent that they colour the clinical picture sufficiently to lead to the supposition that the patient is suffering from cerebellar disease of a kind that requires operative interference, instead of which the symptoms are due to a general disease like disseminate sclerosis, in which, as you are well aware, no operation can be of the slightest avail.

Now, from the point of view of practical physicians and surgeons, the two diseases of the cerebellum that concern us most, if we exclude the general disease disseminate sclerosis, in which the cerebellum has only a part interest, are tumours and abscesses. My object this afternoon will not be to discuss in detail the clinical picture which an abscess may occasion as contrasted with that which may be caused by a tumour, or to contrast the symptoms of an abscess in the cerebellum with those of one in the temporo-sphenoidal lobe, or of tumours in these situations or elsewhere, but rather to sketch for you as best I can the clinical picture that you should look for when there is a gross lesion of the cerebellum, such as a tumour or an abscess which leads to cerebellar insufficiency. I say a very definite clinical picture may result, and my object will be to attempt to portray that picture to you in order that you may be able to recognise for yourselves the condition if cases of the kind come under your observation.

To begin with, of course a case of intra-cranial tumour—it will be largely in connection with intra-cranial tumours that I shall have to deal—when situated in the cerebellum gives rise to certain general manifestations which are common to all intra-cranial tumours irrespective of the position which they occupy. There is headache, paroxysmal in character, such as may occur in any case of tumour inside the skull. In addition to the fact that there is headache the position of the pain must be noted. In cerebellar tumours the headache is very often occipital, and moreover the pain may be more severe on the side of the tumour when the growth is unilateral. In this way information may be derived from the headache as to the possible site of the tumour. Remember, however, that in some cases, instead of the pain being most severe at the back of the head, it may be referred to the opposite temporal region, the opposite fronto-temporal region; there may be a fixed boring pain there, rather than in the occipital region, where you would be more likely to expect it. More important than the pain in the localisation of a tumour is tenderness, and here again in cerebellar tumours help may be derived from local tenderness, on pressure in the occipital region. More important than local tenderness is local bulging, if that be present. Now, it is notably in children that this sign comes to our aid in the localisation of intra-cranial tumours, and it is best to have the head shaved when you are looking for bulging. With the head shaved any prominence on one side in the occipital region as compared with the other is more likely to be evident.

Passing from the question of headache, and having referred to tenderness and local bulging, we next consider another symptom which is common to all cases of intra-cranial tumour, viz. vomiting. Now, vomiting is especially common and especially severe in cerebellar tumours. You know that vomiting may be very severe in any case of intra-cranial tumour, but it is especially common and severe if the tumour is in the cerebellum. The same may be said of optic neuritis. Optic neuritis may be absent in cases

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of cerebellar tumour, but far more commonly we find that optic neuritis is early, that it becomes severe with much swelling, is attended with multiple hæmorrhages, and that it goes on rapidly to atrophy of the optic nerves unless the condition be relieved by operative intervention. The relief of intra-cranial pressure by opening the skull and dura mater may stop the optic neuritis, but unless this is done the patient is certain to develop optic atrophy. So much for the general symptoms, in addition to which there may be coma from intra-cranial pressure and various effects upon the heart and respiration, and so on, which I do not propose to dwell upon now, except to remind you that patients suffering from such tumours may die suddenly from respiratory failure, and that patients with tumours of the cerebellum commonly die in this way where operation has not been undertaken.

My main object this afternoon, however, is not so much to call your attention to these conditions, which are common to all forms of intra-cranial tumour, but rather to direct your attention to the clinical picture that results from cerebellar insufficiency consequent upon gross lesions that destroy the organ. Both from the experimental and from the clinical standpoints these cases have been studied most carefully. Observers are not by any means agreed with regard to all of the results, however, so that what I am going to tell you this afternoon is based largely upon one's own personal experience, both from the experimental and clinical sides of the question. This experience is, however, supported by the results of a large number of cases that have been carefully studied in this hospital before and after operative intervention. A most important paper on the subject has been published in 'Brain' by Dr. Gordon Holmes and Dr. Grainger Stewart, giving the results of their observations, a paper which is particularly important because these observers have been able to study cases of gross lesions of the cerebellum both before and after operation. The process of removal of the disease from the cerebellum of course leads to an increase in the symptoms of cerebellar insufficiency for a time. The result is that a careful study of such cases immediately after operation has given Dr. Holmes and Dr. Stewart opportunities which are most important, for they have thus been able to study in man the phenomena which physiologists have to study in animals, and man can tell us things which animals cannot. Man can tell us what his sensations are, his experiences in regard to what he feels, which sensations are most important, as I shall be able to show you when I have two patients brought into the room presently, with whose cases we shall chiefly deal this afternoon. One of the patients presented from the commencement the attitude—which is the first thing I want to deal with—the attitude that may be assumed by a patient suffering from cerebellar disease. First, we have the attitude of the head, next the position of the trunk, and then the way in which the patient stands. As regards the head, the attitude assumed in cases of cerebellar disease is one in which there is an inclination of the head to the side of the lesion, so that the ear on the affected side becomes approximated to the shoulder on that side, but the chin is directed to the opposite side. Let us assume that this patient has a left-sided cerebellar insufficiency from ablation or from tumour-destruction, or anything of the kind, then the head will be inclined with the ear towards the left shoulder and with the chin pointing in the opposite direction. This patient now before you has not manifested that sign, but the patient you are to see next did. Both of the patients had a tumour on the left side of the cerebellum, the first inside the cerebellum and the other outside of it, growing from the auditory nerve. In the removal of the tumour in the second case the left lobe of the cerebellum had to be destroyed in order to get the tumour properly away; therefore both these patients had destruction of the left lateral lobes, and in that way both proved good subjects for studying the manifestations that result when there is an insufficiency of the left side of the cerebellum. The second man had the cerebellar attitude of his head. Now as to the position of the trunk. The condition is not so commonly seen in man, but consists in curving of the trunk with the concavity towards the side of the lesion. Then as to the lower limbs, the patients require a wider base than normal persons in consequence of inco-ordination, so that they stand with the feet wider apart than is usual.

Next, as a consequence of the destruction of the cerebellum there is, as you know, inco-ordination. Now, the inco-ordination may be manifest while

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the individual is still lying in bed, and may be evidenced by a tendency to rotate about the long axis. This tendency to rotation may, of course, result in the subjects of experimental ablation of the organ, and it may also be present in man. This first patient presented this phenomenon. With his left cerebellar insufficiency his tendency was to rotate with his left shoulder advancing, or like a right-handed screw entering an object. Next as to their sensations. In both of the patients there was the same defect, a feeling that they were being dragged over to one side. This was to the side of the lesion in both cases. This feeling of being dragged over was accompanied by external manifestation of the inco-ordination as evidenced by the mode of the patients' progression. You may see this by observing the way in which this man walks. He described his difficulty somewhat as follows: that when he attempted to walk he would begin in a straight line and then he would be gradually drawn to the left, so that he departed from the straight line, and, having recognised that he had done so he would bring himself back to the proper line by an effort. The first patient will show you for himself what he used to do. He is no longer impelled to walk in this way, but he had the defect so impressed upon him when he was ill that he is able to show you what used to happen, and you will agree that he is a good actor. He will afterwards show you how he can now walk. You see that he is just a little unsteady, but nothing more. Note in this connection that there is an extraordinary provision for compensation of cerebellar defects. It is found to be the same from both experimental and clinical standpoints; that the cerebellum is an organ in which destructive lesions give rise to the most pronounced disturbances of function, but that compensation comes about in the most extraordinarily rapid way, so that after ablation experiments there may be the most perfect return to the normal, and with clinical conditions with the most pronounced inco-ordination such as was present in this case, the patient may recover to the extent you have seen in this man. The second patient has not had so long in which to recover, and you can see that his gait is still defective. Observe the position of his legs, the inco-ordination of the left limb, and the tendency for him to incline to one side in walking and to fall to that side. This defect is not due to his left leg giving way; he does not fall because of insufficient power to maintain the erect posture in consequence of paralysis of the leg, but owing to inability to maintain his balance. The inco-ordinate gait is like that of an inebriated person, as was evidenced by the way the first man walked, and the tendency is for the patients to feel that they are being dragged to the side of the lesion, and for them to fall in that direction. The reason I have impressed upon you that there is this loss of balance is owing to the fact that Luciani more especially, in his experimental observations, has pointed out a loss of power on the same side, so that some of the tendency to fall to the same side has been ascribed to that cause. In this man we have proof that the tendency to fall is due to an abnormal sensation which the patient does his best to overcome, and that it is not due to weakness of the lower limb on the side to which he falls. Similarly, if you make these patients stand with the feet together and eyes closed you find that they will always tend to fall in the same direction as that in which they tend to reel.

Next in connection with inco-ordination we have to study the condition of the arms. Both these men have recovered from the inco-ordination which they presented in the arms; but remember that patients with cerebellar defects may show marked inco-ordination in the upper limb on the side of the lesion. There may be some inco-ordination of the opposite upper limb also, but the defect is usually most evident in the upper limb on the same side as the lesion. Recovery has been very good in these men, so there is very little defect to be seen now. This man is not quite accurate in touching objects aimed at, but there is practically very little wrong. There is a slight difference on the two sides, but nothing pronounced, whereas before the difference was very striking. There is no difference of opinion as regards inco-ordination; everybody is agreed that with lesions of the cerebellum this defect results.

I want now to call your attention to the question of motor weakness in the limbs on the same side as the lesion. Here experimental physiologists and clinicians are at variance. There are those who maintain that all the motor disability which is met with in cerebellar lesions, whether experimental or as the result of disease, is consequent upon

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inco-ordination. That is a position which I am unable to support. On the contrary, I most strongly agree with those who are of opinion that as a consequence of experimental ablation and as a result of destructive lesions of the cerebellum in man definite motor weakness occurs. The weakness is on the homolateral side—that is to say, on the side of the lesion—and it is a definite loss of motor power quite distinct from the inco-ordination which also results. Now, this motor weakness was determined in both these men; both of them had marked weakness in the arm and leg on the left side. This is a condition which is brought out very clearly in the paper by Dr. Holmes and Dr. Stewart to which I have already referred. In many cases where careful tests are applied definite motor weakness may be determined. Note that, in contrast to disease of the brain, which occasions hemiplegic weakness on the opposite side of the body to the side of the brain which is affected, lesions of the cerebellum supply us with motor insufficiency on the same side as the lesion. The condition differs from cerebral paralysis in another way. You are prepared to find spastic paralysis when motor weakness, hemiplegic in distribution, is due to a lesion of the brain, but when a lesion of the cerebellum occasions motor weakness, it is a flaccid form of paralysis that results. The first man had the most flail-like condition of his limbs, and even now his muscles have not recovered their normal tone. His limbs were never spastic and they are still too limp. There is thus a condition of flaccidity as opposed to the spastic weakness that is more usual in cerebral paralysis. Furthermore, the hemiplegic weakness that results in cerebellar disease is not shared by the face. The limbs are affected but the face escapes. Some of you can see in this patient very marked facial paralysis on the left side, and you may therefore possibly think that I am talking at random, but I am not: I am speaking quite deliberately when I say that as a consequence of cerebellar defect you may have motor weakness in the limbs on the same side, but that you do not find any weakness in the face. The face may, however, suffer in cases of gross lesions of the cerebellum, such as tumours or abscesses, in consequence of pressure on the facial nerve. Pressure on the facial nerve may give rise to facial paralysis, which differs from that which results from a brain-lesion, notably in the fact that the upper and lower face become affected in equal degree, and that the reaction of degeneration may be observed on electrical examination of the facial muscles. Moreover the tongue does not participate in the paralysis as it usually does when there is a lesion of the brain. You know that when a lesion of the brain causes facial paralysis the tongue is usually paralysed with the face, so that it deviates to the paralysed side when protruded. In these cases, on the other hand, it is a facial paralysis consequent on pressure on the facial nerve, in virtue of which the face is affected without the tongue. The first of these men presented only slight facial weakness, from which he has practically completely recovered. It was never very marked but was quite definite. The second man has had complete destruction of his facial nerve, with complete reaction of degeneration in the muscles, and if you look at him you will see that when he attempts to close his eyes the only thing that happens is that his eyeball rolls up under the upper lid, and all other attempts at movement reveal complete paralysis of the face on that side. This is in consequence of pressure on the facial nerve, and is not due to any defect of the cerebellum.

While dealing with motor defect in connection with the facial nerve, one has to call your attention to the fact that in some of these cases there may be pressure on the fifth nerve, with, as a consequence, motor and sensory paralysis. This second patient, although he has sensory paralysis of the fifth, has no motor paralysis. His jaw comes down mesially when he opens his mouth, even against resistance, and his masseters and temporals act equally on the two sides.

Paralysis of the ocular muscles may also result in these cases of cerebellar tumour, owing to pressure on the ocular nerves. It is notably the sixth nerve which becomes affected, and in both of these patients this was the case. Weakness of the external rectus on the same side resulted in both, owing to pressure on the sixth nerve. Unfortunately, gentlemen, we cannot lay much stress on the sixth nerve paralysis as an aid in localisation, because this nerve so commonly suffers in cases of increased intra-cranial pressure of tumours of all sorts and in all situations. We cannot therefore say that because there is paralysis or paresis of the external rectus this of necessity is a point

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of much value in localisation. When, however, the sixth nerve is paralysed in conjunction with others, as, for instance, the facial or fifth on the same side, then of course it shares with them their value in localising the lesion. The reason I have given you, however, robs it of value when paralysis of the sixth nerve stands out as an isolated sign, unsupported by paralysis of any other cranial nerves on the same side.

Reference to the motor disability which may result from pressure on the sixth nerve leads me to call your attention to the fact that although no motor paresis of the face results as a direct consequence of insufficiency of the cerebellum itself, there may be evidence of motor weakness in connection with the ocular movements as a consequence of cerebellar defects. There may be weakness which I have referred to as a consequence of pressure on the sixth nerve, but apart from this remember that as a result of cerebellar insufficiency there may be definite motor weakness, leading to strabismus and to defects in the movements of the globes. The first point I wish to make clear in this connection is that what has been called "skew deviation" may result from an acute lesion of the cerebellum. This condition existed in this man, as is shown by the photograph which I shall pass round for you to see. Observe that one eye is turned inwards and downwards and the other eye upwards and slightly outwards. It happens that this is the position which the globes assume in the majority of cases in man; that is to say that, given a left-sided cerebellar deficiency, the left eye turns down and in and the right eye up and out. This, however, is not what I have found on experimental ablation of half of the cerebellum in the laboratory. There a different state of things was observed, for the opposite eye turned down and out, and the eye on the same side, instead of turning down and in, was directed up and out. In clinical cases we find, as Dr. Holmes and Dr. Stewart point out in their paper, the eyes turn down and in on the side of the lesion and up and out on the opposite side. It is not of much importance that there is this slight difference in regard to the experimental and the clinical results, for the skew deviation tends to pass off pretty rapidly in both. Then, however, what is noticed most is that the patients—and the same obtains in laboratory investigations—have a difficulty in moving the eyes to the side of the lesion. In other words, there is some weakness of the conjugate movement. This is quite distinct from any paralysis due to pressure on the sixth nerve, because the opposite internal rectus may show similar inability to move that eye properly towards the side of the lesion. While dealing with these motor defects in connection with the ocular movements, reference must be made to nystagmus, because this phenomenon is most important. You know that everybody is agreed as to the importance of nystagmus in the diagnosis of cerebellar disease, but what I want specially to insist upon is the importance of the direction of the nystagmus and its character as aids in localisation. It seems clear that, at any rate in the large proportion of clinical cases, and certainly constantly from the experimental standpoint, the eyes tend to deviate from the side of the lesion, and there is difficulty in moving them towards the lesion, and that the nystagmus which results on turning the eyes from the lesion is less marked, and if pronounced is usually much quicker than the nystagmus seen when the patient attempts to turn the eyes to the side of the lesion. In the latter case the nystagmus is usually more marked and much coarser. The second patient illustrates this, for he has a little quick nystagmus on turning the eyes towards the right and a much coarser nystagmus when he turns them to the left. The first patient's nystagmus has almost disappeared, but what there is is more marked when he looks to the left than when he turns his eyes to the right. These are the only motor defects that I wish to call your attention to in connection with diseases of the cerebellum.

Next turn your attention to the question of sensory defects as a consequence of cerebellar lesions. It is a very difficult matter indeed to be certain of slight defects of sensibility as a result of experimental lesions in the laboratory, so that one has to be very careful not to be dogmatic on the subject. It did, however, seem fairly clear that there was in the subjects of experimentation a homolateral anæsthesia—that is to say, a defect of sensibility on the same side, and also some defect in one limb on the opposite side, but notably defect in the limbs on the same side as the lesion. Nothing of the kind has, however, been observed in man, and none has been elicited on careful testing in these

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patients, so that from the clinical standpoint cerebellar disease responsible for cerebellar insufficiency does not occasion as part of the clinical picture any anæsthesia of the limbs on the affected side. We may have motor paresis and inco-ordination, but no anæsthesia of the limbs. There may, however, be anæsthesia of the face, but this depends on pressure on the fifth nerve, a condition of things which exists in this second patient. He has return of sensibility now in so far that he recognises touches on the left side of the face, but he still has analgesia, for he does not recognise painful impressions. At one time he could not detect any form of stimulus on the left side of the face. Remember, however, that this defect is not in any way due to cerebellar insufficiency, but is the result of pressure on the fifth nerve.

I must now say a word or two about the special senses. In cases of increased intra-cranial pressure there are disturbances of smell, as you know, and therefore in cases of cerebellar tumour, as in any case of great increase of intra-cranial pressure, anosmia may result; but this cannot be regarded as of any special significance when search for signs of cerebellar disease is being made. We also derive no assistance from the sense of taste.

You have gathered from what was said when optic neuritis and blindness were discussed that, of course, sight may be markedly affected when the disease is in the cerebellum. This, again, however, is something which does not supply us with any special information in regard to the position of the disease causing the optic neuritis which leads to the blindness. It is otherwise with regard to the other special sense to which we have yet to refer, namely hearing. Hearing is all-important in connection with these cerebellar cases, not because of the defect in the cerebellum itself, but because of pressure on the auditory nerve. You know that the facial and the auditory nerves lie side by side at the base of the brain, and you know that these two nerves often suffer together. With various lesions at the base of the brain which cause paralysis of the face owing to pressure on the facial nerve, it is common to have the auditory nerve destroyed also, and, in consequence, deafness results on the same side as the facial paralysis. Now, gentlemen, you might think that deafness would be the more common of the two if only one of these defects resulted, the portio mollis suffering more than the other nerve. In reality, however, many of these cases of cerebellar disease present motor weakness of the face when there is no defect of hearing. This first patient has no defect of hearing and never has had, although he had motor paralysis on the left side of his face. It is not fair to ask you to believe this, because he has recovered so completely from the facial paralysis that you can no longer detect any such defect. Nevertheless, I repeat, he has never had any difficulty with hearing, although he has had facial weakness. This other patient is, however, absolutely deaf on the left side. He can hear my watch perfectly on the right side but not on the left. Defect or loss of hearing is accordingly the most important evidence which can be obtained from the special senses in the search for signs that can be regarded as of value in the localisation of tumours of the cerebellum. Moreover, affection of the auditory nerve, like affection of the fifth and of the seventh, is most likely to result in cases of extra-medullary tumours of the cerebellum, so that deafness is a most important aid in attempts to localise the exact position of a tumour.

I must now ask you to turn your attention to the state of the tendon-jerks. Do we gain any information from them in our attempts to localise gross lesions of the cerebellum? As regards the knee-jerks, all that we can say is that we do not obtain any positive information that can be considered reliable. Experimentally all is plain sailing, for there is exaggeration of the tendon-jerks, with an increase more especially on the side of the lesion. As time goes on and recovery results, the knee-jerks subside in their activity, but with this change the knee-jerk on the affected side still continues more active than on the other side. This is, however, not the case in man. We find great variations in the state of the knee-jerks; sometimes they are exaggerated, sometimes lost, and they come and go in an irregular fashion. The general tendency has, however, been that where cases have been studied after operations an increase of knee-jerks has been met with, and the knee-jerk on the side of the lesion is the one that has been the more active. When this first patient came into the hospital he had lost his knee-jerks, whereas the other patient has had exaggerated knee-jerks all the time. The first patient's knee-jerks have returned, while the other patient, whose knee-jerks are exaggerated, has a more active jerk on the side of the lesion than on the other side, which is the one point that may be of assistance in localisation. The knee-jerks are, however, so variable in man that it cannot be said that they are of much assistance in localising cerebellar lesions.

Now as to ankle-clonus. I have no personal

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experience of ankle-clonus as a consequence of cerebellar lesions in man; but in the paper to which I have more than once referred Dr. Holmes and Dr. Stewart state that in several instances ankle-clonus has been present after lesions produced in the removal of tumours of the cerebellum. There is no spasticity, you will remember: it is a flaccid paralysis, and yet exaggerated knee-jerks and ankle-clonus are present, and the ankle-clonus has been on the side of the lesion. Ankle-clonus has been present in cases after operation, but in none before operation.

I ought to conclude as the time at my disposal has expired, but I hope you will allow me to refer to one or two more points. What have we to say of the plantar reflex, about which we make so much in the localisation of cerebral lesions? The plantar reflex does not appear to be affected by lesions of the cerebellum—that is to say as a direct consequence of lesions of this part of the nervous system. Thus we do not find the extensor response as a consequence of cerebellar defects in the way in which ankle-clonus and exaggeration of the knee-jerks have been found after operations on the cerebellum. The plantar reflex may, however, be of the extensor type. When it has been so, if we leave out of account such cases as tumours of the cerebellum which invade the pons or press upon it, and confine our attention to cases of cerebellar tumours in which there is no such interference with the pons, the extensor response has only been found where there has been hydrocephalus. We know that any case of hydrocephalus, no matter what its cause, may be responsible for the extensor type of plantar reflex in consequence of disturbance of the cortex or some other part of the pyramidal system. The same naturally obtains when the hydrocephalus is the outcome of disease of the cerebellum. It is not, therefore, a direct result of the cerebellar defect, but is a secondary consequence of the hydrocephalus induced by the tumour of the cerebellum.

Finally, there is nothing to be noted with regard to the sphincters, except that they escape in the case of cerebellar lesions, just as they do in the large majority of cases of lesions of the brain.

In concluding these remarks that I have had to offer to you in regard to the clinical picture that may result from gross lesions of the cerebellum, it is fitting that a tribute should be paid to our surgeons. The first of the two patients you have seen this afternoon was in a comatose condition, and his case had been given up as hopeless, when his mother brought him to this hospital. So urgent was his condition that he was operated on the day after his admission, and yet here he is to-day quite well—a splendid example of the triumphs of modern surgery!

August 6th, 1906.

FRACTURE IN THE INSANE.*

By J. F. BRISCOE, M.R.C.S.

GENTLEMEN,—It is believed by many that the insane are very liable to fractures. This popular belief appears, however, to be based on hearsay evidence. Some time ago at Winchester I read an account of a remarkable case of fracture in the insane which terminated most successfully, and in 1898 I discussed the osseous system of the insane before a meeting in London of the Psychological Association of Great Britain and Ireland, illustrating my paper by a collection of bones of sane and of insane persons. At this meeting a discussion followed, of which I give you a brief abstract. One gentleman remarked: "I am clear about this, that an infinite deal of nonsense has been talked upon the subject, because when lunatics die with broken ribs the medical opinion given is usually to the effect that the bones of lunatics generally, and in the abstract, are fragile." Whereas the question is not, "Are the bones of all lunatics fragile?" but "Are these bones fragile?" He also stated that "it depends more upon the general physical condition of the patient than upon his mental state." Again, this same authority remarked that he had had "two patients killed by their ribs having been broken." Another psychologist had noticed at post mortem an old fractured rib united with fibrous capsule, probably of some months' duration. And a third debater thought it extraordinary that the theory about the softening and fragility of the bones of the insane occurs in reference to the ribs and sternum only. He had also met with few fractures amongst cases of general paralysis, and thought that broken limb bones were not more frequent in general paralysis than in other cases of mental disease. A fourth speaker differed with the last, remarking that "there could be no question as to the relative frequency with which one could easily break a lunatic's ribs, especially in general paralysis of the insane." The same alienist stated that "it would be absolutely impossible to establish that there is not excessive softness, not, perhaps, always to breaking point, but to bending point, in the post-

* Remarks delivered at Southampton before the Southern Branch of the British Medical Association.

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mortem room." But his concluding statement speaks for itself when he says: "I have heard of a case in which it was suggested that the breaking of several ribs was due to auscultation."

In the debate from which I quote there are three other speakers to mention. One said: "I think it has been established beyond all doubt that the bones of old people are soft, and in certain cases of general paralysis they are very much softened; it is a question, too, whether these fractures do not occasionally occur spontaneously." Another stated he had "seen a case of fracture in an epileptic seizure from muscular spasm." And the last speaker observed he had witnessed "softening of the bones of the male and female general paralytic without fracture," but he added, "It is more often seen in senile cases."

"Are fractures common in your asylum, and do they do well?" This is a question which I have raised and forwarded in a printed circular to no less than seventy medical superintendents of public and private asylums of the British Isles. I have also incorporated in this circular questions referring to post-mortem evidence of disease of the bones in the insane, the opinion often expressed regarding the tendency to softening of the bones of the general paralytic, and the terms "brittleness" and "softening," which are used when discussing the osseous system of the insane.

The British Isles contain a large area of ground covered by lunatic asylums. England and Wales maintain the highest number of asylums, Scotland next, and Ireland figures last. In these countries I learn, in answer to my circular-letter, that the insane there resident do not often break their bones, and when they do so the results are invariably good. In attempting to convince you of this fact I cannot draw up a complete statistical table referring to the particular asylums whence I have gleaned my information, for this would be committing a breach of confidence; yet I can discuss the direct written evidence of the medical superintendents of these asylums with you without mentioning their names, and you will get a fair idea of what I have gathered and endeavoured to compile truthfully. One medical superintendent informs me that he has had only one case of fracture in eight years and that it did well; this in a large private asylum. A second has had none; this also a fairly large private asylum. From an idiot asylum only two cases are reported in three years. Another medical superintendent of a well-known large public asylum says, "We have not had a fracture for years." In a Scotch asylum thirteen fractures have occurred during three years, among an average of 430 patients; all without exception have done well. From another Scotch asylum the medical superintendent says: "Not common, and they do notoriously well, but the utmost care and local effective treatment is essential." An Irish medical superintendent remarks: "It is a matter of opinion whether fractures are common, but I consider them not frequent considering the overcrowding and population (1700)—about seven patients annually." And from another Irish asylum the medical superintendent says: "No, they are not common, and do well, except cases of the neck of the femur." And with regard to this latter point I find that it is these senile cases of fractures that give rise to trouble in other asylums. A Scotch asylum, with a population of 750, has had seven cases of fracture of bones during the last five years, and in another Scotch institution, where they employ female nurses in the male wards, there have been in three years, with a population of 550 patients, fifteen instances of fractures (including such minor cases as fracture of metacarpal bones, etc.).

Here is an example of a case in another asylum from Ireland. A general paralytic: syphilis, Pott's fracture, with repair ten weeks before death. A male, æt. 40 years, admitted into the asylum from a workhouse in an advanced stage of general paralysis. Eighteen days after admission he had a seizure, followed by monoparesis and acute emotional outbreaks. He died ten weeks from the date of the fracture. The case was published in the 'Journal of Mental Science' by Dr. M. J. Nolan, January, 1904. From a surgical point of view it is instructive; for although the patient had syphilis and general paralysis in the last stage, the fracture was completely repaired, for he used the limb, getting out of bed and placing his entire weight upon it. Medico-legally speaking, it is more interesting to psychologists, because, as Dr. M. J. Nolan says, "it is obvious the accident did not accelerate death." During four years, with a population of 500, I get a report of ten fractures from a reliable Scotch authority; and from an English asylum of 300 patients one per annum, while from a well-regulated hospital asylum the medical

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superintendent says: "Not common. I do not think they occur in larger proportion than in the sane population. Most have done well." And in supporting this opinion I think, too, if we were to draw out a table we should find that below the age of forty fractures in the insane per centum were considerably lower than in the sane. In fact, some asylums give evidence that, taken altogether, fractures are more common with the sane than with the insane. Finally, with one exception, the majority of the medical superintendents who have replied to the question "Are fractures common in your asylum?" speak of their rarity. A few say they are infrequent. One thinks the word "common" suggests culpability, but he says: "Fractures do occur from time to time, and the result depends partly upon the nature of the fracture and partly upon the mental and physical conditions of the patient." The rest of the medical superintendents say: "They are uncommon. Moreover, they all seem to do well, unless there is some complication with great physical illness." So the insane mind does not appear to affect materially the reparative process of bone. And that they should do well under treatment is inevitable, considering the few that come under treatment (according to the replies of my circular-letter) and the anxious care with which these patients have to be watched and attended to. Nevertheless, admitting that fractures in the insane may be infrequent and that they do well, there must still be cases in asylums which, like similar cases outside these institutions, are difficult to account for and difficult to cure, however watchful the medical superintendent may be. In Westbrooke House, a private asylum, and a small institution compared with a public asylum, in which the well-to-do have their home-like surroundings, one would expect fractures to be very infrequent because the patients have individual attention. Obviously it is to the best interests of similar legalised institutions that the patients should not break their bones or in any other way fall into mischief. Moreover, being supplied with selected mental nurses, the patients ought to be better off than in the public asylums, where individual attention is an impossibility. Thus it would seem more momentous to have a fracture in a private than in a public asylum. In the above-mentioned home, licensed for thirty patients, with varying admissions, discharges and deaths, there are recorded in ten years three cases of fracture, the first two of which were slight. The third case was a lady, aged 52 years, who while dressing her hair fell suddenly to the ground and fractured both legs. It has been since proved that this was due to petit mal. The upper fractured end of the left tibia was riding well forward, two or three inches through the stocking and a fair amount of blood escaped. The tissues over the end of the right leg bones were very thin. For obvious reasons I handed the case over to my professional friends Drs. Leslie and Williams of Alton. The wound on the left leg was brought together by sutures, but on the fifth day there was redness, swelling, heat, pain, and pus-formation. Again, the skin was very tense over the sutures, and the upper fragment, considerably interrupted out of the line of continuity, was found to be irreducible. Counter pads, padded small splints, raising the heel, and every known device we could think of, failed to get any approximation of the fractured ends. The temperature, too, began to rise, although so far it was only 101° F. Still, time was valuable if the left limb was to be saved; so we secured the services of Mr. W. Arbuthnot Lane and Colonel Rainsford, R.A.M.C., of the Princess Louise Hospital, Alton, and Mr. Lane wired the tibia. The case was not so protracted as is usual with these double and compound fractures of the legs as described in surgical works. Our strained efforts were undoubtedly crowned with success beyond expectation, by means of silver wire, and within the recognised period for the treatment of compound fractures of the legs. The result appears to be an instructive example of skill and ingenuity in surgery. Skiagraphy has certainly proved this to be a fact, and this has been confirmed, too, by outward measurement of both legs. The unwired leg measures about one inch shorter than the wired leg. The insane lady says she can walk as well now as before the accident. And this seems to be the case, although she stands on the one leg, the wired one, more comfortably than on the other. I have danced with her since the catastrophe, so that speaks for itself.

There is no reason why the rib-bone should be considered apart from the other bones of the body, but in the insane it is perhaps more often exposed to risks of injury than any other bone in the body, and owing to its thinness yields more readily. That

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it has a distinct vitalisation apart from the rest of the skeleton is not proved. The intercostal arteries arise direct from the aorta, and owing to a diseased state of the main vessel, the symptoms of which are not recognised during life, the blood-supply may become directly interfered with, and thus affect the proper nourishment of the bone. This, of course, is only a theoretical expression of opinion, yet I am not aware that the intercostal arteries in particular have had their due share of attention from the hands of the pathologist. But we do know this, that "a diseased coronary artery is not always to the best interests of the heart." It is, however, remarkable that only with the insane do we hear of these softened ribs apart from the developmental disease rickets or the acquired affection mollities ossium. I have never heard it stated that cancer or tuberculosis produces "brittleness" or "softening" of the bones unless these diseases directly attack the bone. When I was studying the subject of cancer many years ago I do not remember undue friableness at post mortem; but then, does the pathologist often consider this matter of the bones at an ordinary autopsy?

The inorganic building up of the bone with lime-salts is apparently as simple a process as the separation of these same salts by means of an acid reagent. It would appear to be purely mechanical. And it is easy to conceive how a badly constructed substratum, or any irritant matter flowing in the neighbourhood, might interfere with this physical action. The friableness of a bone would seem to depend upon (*a*) the condition of the substratum of the bone, (*b*) the physico-chemical state of the fluid circulating in the vascular system of the bone, (*c*) the physical state of the vascular system of the person. The pathology of rickets and the disease of bone called "mollities ossium" come under these headings. The former is common enough, and in the majority of cases yields readily to dietetic and suitable regimen, while the latter seems rather an intractable complaint, and is much more often observed in some parts of the world than in others.

Now I come to the second question in my printed circular to asylum medical superintendents—"Do you often find at post mortems distinct evidence of disease of the bones in the insane, either brittleness or softening?" In examining the answers to this question I find fifteen in the affirmative and eighteen in the negative; and all these medical superintendents state that it is rare. Others are unable to vote since they seldom do post mortems and some do not do enough to hazard an opinion. One says in answer to the question: "I have done some hundreds of post mortems in the insane, and have rarely found undue brittleness and softening, but such cases certainly occur." Dr. Caldecott, of the Earlswood Idiot Asylum, tells me that he often finds cartilage of ribs ossified a little prematurely.

Other questions in my circular were as follows: "What is your opinion regarding the statement that general paralytics have a tendency to softening of the ribs?" "Do you ever find this condition in the bones of other forms of mental disease?" "Besides this softening of the ribs in the general paralytic have you noticed the rest of their bones thus affected?" In giving you an analysis of fifty answers I cannot do better than classify them as follows: Yes, 23. No, 4. Some, 1. No experience, 5. No post mortems in these asylums, 2. No answer to the question, 10. No evidence, 1. Rare, not apart from physical disease, 1. No general paralytics in these asylums, 3. Of the 23 who maintain very firmly that the bones are softened in the insane 11 are from English asylums, 6 from Scotland, 4 Irish, and 2 Welsh. And in selecting from these reports what I consider the best, I do so from those whose statements would, I think, be respected and valued by the Psychological Association. One of these, an authority on general paralysis, writes as follows: "Ribs and other bones softened in general paralysis, also occasionally in secondary dements, of course in some patients with marked mollities ossium more or less generalised."

But I should like to ask this authority, "Is not mollities ossium rare nowadays in asylums for the insane?" and that when it appeared to be common (*vide* 'Edinburgh Medical Journal,' August, 1862) "was it not encouraged by indifferent management and indifferent feeding?" Such cases in particular as are recorded in the above journal arose probably from specific influences operating through the blood plasma.

Another Scotch alienist remarks: "I think that the statement that general paralytics have a tendency to softening of the ribs is an erroneous one—all persons who take no exercise and are spoon-fed

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have weak bones. I have never found anything special in the bones of the mentally afflicted." But the answer of an equally sound Scotch opinion is the following: "The majority of general paralytics have atrophied and softened ribs. Many, however, have atrophied and brittle ribs, while a number have quite normal development and strength of ribs." And here is an equally valuable opinion from Scotland: "Yes, I agree, but in my experience the other bones (*e.g.* femur, humerus) are as liable as the ribs. Yes, and in senile insanity. The most striking case I ever saw was in a woman suffering from chronic delusional insanity and acute miliary tuberculosis." The next reply is from an English asylum, and it is as follows: "There appears to me to be a softening of all the bones in general paralysis—a part of the degenerative changes which, in my opinion, the whole organism is undergoing." Another observer has noticed that "occasionally the sternum is affected as well as the ribs." And, to finish my report on the fifty replies, I cannot withhold two answers, one from the resident physician of a standard English asylum and the other from the pathologist of an equally well-known English asylum. The former makes the following written statement to me: "Brittleness and softening of the bones of the insane here are associated only with concurrent physical diseases—*e.g.* chronic diseases, septic conditions. Cases of brittleness of the ribs in general paralysis are, in my experience, quite rare." And with regard to softening of the bones "the condition is quite rare, and is not apart from associated physical disease." The latter writes in a style which is indicative of his close acquaintance with the post-mortem room, and he remarks: "General paralytics have a tendency to so-called 'softening' of the ribs. This condition is found in the bones in other forms of mental disease, but, excluding senile cases, not relatively with so much frequency as it is in general paralytics. Occasionally it has been noticed in other bones besides the ribs, but with the exception of the skull bones, other bones are not systematically examined (for obvious reasons) except in special cases."

A great deal has been written and remarked upon the friable bones of the insane by means of trophic influence, and again of the neurotrophic diseases of tissues in general. A well-known System of Medicine states the following: "Mental shock may turn the hair white, and it has been followed by pigmentary changes in the skin itself. Reflex action has also produced such effects as effusion into joints and affections of the skin. But very little is known definitely of peripheral lesions due to cerebral disease or to reflex action." It is interesting to note that the neurotrophic affections of bones and joints were formulated in 1831, and that in 1869 Dr. Clifford Allbutt was the first in this country to report a case of locomotor ataxy. Then in 1873 Weir Mitchell came forward with spontaneous fracture in tabes; and since these dates there has been a considerable advance with the trophoneuroses, not only of the bones and joints, but of the soft tissues. Again, of recent date, histological changes of gross lesions in the posterior spinal ganglia, the sensory roots, and the peripheral nerves have been contributed as explanatory of brittle bones; and no doubt these are stepping-stones to a closer investigation of the nervous influence on bone. However, it is well to remember that at present there is no proof that the ultimate structure of bone is directly influenced by nervous twigs as are the hair and the softer tissues. But we do know this, that the fine nerves which may be seen entering the bones along with the arteries are probably destined chiefly for those vessels which they perforate to the middle coat, supplying the muscular element of the artery as far as unstriped muscular fibre traverses the minute vascular system. Again, it must be remembered that an arterial vessel is insensitive, like bone, unless it is either unhealthy or inflamed. A remarkable fact in insanity is that gross changes in the cerebral structures are not infrequently observed. But we do know something about the vascular system, and are not our bones as old as our arteries? And is not the vascular system of the bones of as much importance to the pathologist as are the nerves which accompany them? Those of us who saw at the annual meeting of the British Medical Association at Oxford in 1904 the stereoscopic skiagrams of the vascular system surrounding a long bone were, no doubt, struck with this important achievement in X-ray work. And thus, I can conceive that in old people, and in the insane, and in others broken down by physical disease, the degree of brittleness and decay of the osseous system is due as much to vascular degeneration as to nervous influence.

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In continuing the discussion on my circular-letter to asylum superintendents of the British Isles, I now approach the last question, "With regard to fracture in the insane which terms are you inclined to use, 'brittleness' or 'softening'?" In analysing 53 answers I find 19 are in favour of brittleness and 6 of softening; 11 prefer both terms; 10 give no replies; 5 say neither; 1 that there is no evidence; and 1 rarely makes use of terms of palliation. Further, by a process of careful selection I have before me eight reports from English, Scottish, and Irish authorities whose opinions are worthy of the highest respect, because they are well-known writers on psychology, and qualified by long experience. They all agree, except two, in the tendency of general paralytics, chronic demented cases, senile insanity, and chronic insanities to develop brittleness and softening of the bones. Of the two who dissent, one says: "I have never found anything special in the bones of the mentally afflicted." The other admits they may be brittle, but remarks "they are rarely softened." But the terms "brittleness" and "softening" appear to be very elastic in the eyes of some asylum medical superintendents. And even pathological psychologists are very liberally disposed to strain at gnats and swallow camels. One gentleman informs me that he prefers the expressions "hard and brittle," or "soft and breakable," or "somewhat," or "slightly," or "moderately," "markedly," or "extremely." Thus he says "somewhat hard" and "markedly brittle" or "moderately soft" and "extremely brittle." Another well-known writer is content with the simple terms "brittleness" and "softening," or a mixture of both. And I maintain if a bone is brittle call it brittle, if it is soft call it soft, for shades of gradation of brittleness or softening of the osseous system appear to me unnecessarily misleading when we possess no standard bone from which we can draw a comparison. I know of no work on osteology wherein is found the measure of the breaking strain of the bones; thus any deductions we may arrive at on testing the breaking strain of the insane bone must be inconclusive unless we have previously established the normal breaking strain of that particular bone in question.

I do not think it desirable to continue any further the discussion on my circular-letter.

To sum up, then, the nature of fragilitas ossium in the insane comes under the atrophic affections of bone. The morbid process follows the lines of the normal process known as "lacunar absorption." In old age resorption of the bones is termed "senile atrophy." And when the resorption is excessive the remaining bony tissue becomes incapable of withstanding an ordinary strain, and so fractures with great readiness; this condition has been termed "symptomatic and osteopsathyrosis or fragilitas ossium." And besides other forms of atrophy of bone, notably we talk of atrophy from pressure, and not least of those types of bone-atrophy which appear as the sequelæ of nervous diseases, and are named accordingly "neuro-paralytic" and "neuropathic" atrophies (Ziegler). The causes of morbid decalcification (halisteresis ossium) are unknown, according to Ziegler. The presence of lactic acid in the bone-marrow is a theoretical suggestion by some authors, while others attribute the solution of bone to an increased amount of CO_2 in the blood. Eisenhart believes that osteomalacia is the result of the diminished alkalinity of the blood, but von Recklinghausen is probably correct when he regards it as essentially due to some local irritation of the vascular mechanism of the bones. But if it is true that our bones suffer more from changes in the vascular system than from a deranged nervous system, then should not a careful survey of the morbid anatomy of the vascular system be of first importance to the psychologist? And considering, too, that bone is largely composed of inorganic matter, receiving, as far as modern anatomists tell us, little nervous influence, except through the vascular system, cannot we conceive that its breaking strain is dependent upon the goodness of its vascular supply? I can quite understand that if the vascular system is at fault normal physiological brittleness may appear earlier than usual, and that in bedridden people, and in those with senile dementia, and not less in general paralysis, softening of the osseous tissue is likely to occur with indifferent nutrient vessels. The hoops, too, of the thorax must thus suffer in the same way from the bad supply of blood they will receive from vessels communicating with an inelastic atheromatous aorta. And besides local irritation of the vascular system being provocative of osteomalacia, it must be remembered that there is an idiopathic form of fragilitas ossium. This idiopathic form is congenital, develops from some unknown reason in adult life, and appears in different members of the same family. And the tendency to inherit fragilitas ossium may be, like the tendency to inherit weak blood-vessels, as yet not thoroughly investigated. Again, may not the osseous system, like the systems in general, be

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"jerry built," using an unscientific expression? It would seem that the organic substratum of the bone, in conjunction with its hardening matter, the lime-salts, is possessed of some morbid character which manifests itself by abnormal brittleness of the bones. The occasional occurrence of fractured ribs in the insane may be in this way explained. Moreover, ribs are thin bones, they are possessed of a delicate curve, are more or less superficial, and if there should be a struggle they are more likely to give way than any other bones of the body. But a struggle cannot always be avoided, and is at times sought by the patient, whose ribs may be, moreover, as I have intimated, of an inelastic fibre. If there is a death the coroner does not always appear to elicit the truth about these broken ribs, and thus opprobrium follows. Of course, a reliable attendant avoids struggling with a patient single-handed, as far as this is possible. But it does seem to me that the pendulum of the law on mechanical restraint has swung too far in the wrong direction. Personally, I would rather be restrained by a jacket or by padded gloves than by the arms of one or more attendants. There is a dual irritation with an attendant, and it it more likely to lead to serious consequences than with ordinary restraint. Even the best of us are liable to lose our self-control when our muscular sense is brought into play under these very trying circumstances. And I contend that regulation mechanical restraint and padded clothes are less likely to lead to fracture in the insane than the restraint of a male attendant.

It is very important to appreciate the fact that all of us have not the opportunity, much less the desire, of making scientific observations on the bones. Our hours are invariably fully engaged, and we do not often get an occasion of examining the dead bodies beyond the soft tissues, unless once in a way. Yet, because in our asylum observations on the bones are infrequent, there is no reason why in another asylum they may not be more frequent. And this induces me to remark that fractures from brittleness and softening may be produced after death during the removal of the sternum, as is evidenced by certain facts: (*a*) the tissues immediately surrounding the fracture are not found injured or swollen; (*b*) there is no adjacent inflammatory action; (*c*) there is no associated healing or reparative process going on; (*d*) a careful examination does not disclose any signs of extravasation of blood or of bruising of tissue, which are almost invariably found at the site of fractures where they have been recently produced—in fact, a complete absence of the appearances which point to fracture having been inflicted during life. Finally, if autopsies are as rare as fractures, is it not likely that something pathological may escape our attention?

August 6th, 1906.

THE PATHOLOGY OF CHILL.

By W. RINGROSE GORE, M.B., B.Ch.

ALL diseases due to bacterial infection can be conveniently divided into two classes, those due to infection from without by bacteria which at no time normally exist in the human body, and those diseases which are due to growth of bacteria which are normal inhabitants of the body in health.

This division is one of extreme importance, as the difference between these two classes is very marked both in the acquirement and course of the diseases in each, perhaps the most marked difference between the two classes of diseases being that an attack of disease caused by infection from without usually confers immunity from further attacks, as in measles, while disease caused by infection from within, instead of giving immunity from further attacks, gives increased liability, as in pneumonia or nasal catarrh.

In this country by far the largest number of diseases and the largest adult death rate are due to the second class of bacterial disease, because in this class are included all those acute diseases due to chill, and the pathology of chill is the pathology of those diseases which are due to the growth of the bacteria normally present in the body. How this growth takes place will be shown by my experiments into the growth of bacteria in nasal catarrh.

The pathology of chill has never been properly worked out, and, in consequence, in diseases due to this cause more and more prominence is being given to the bacterial factor, to the neglect of the really more important factor of chill; for while we can to some extent prevent chill, we can never get rid of those bacteria whose growth is allowed by the changes in their environment that chill produces.

The first essential of a chill disease is that the bacteria which cause the disease must be present in the body when the disease is induced. If you get pneumonia by getting wet through, you do not find the pneumococcus in your wet clothes. Unless it was present in the body the chill caused by the wetting could not result in pneumonia, so that a chill disease due to bacteria necessarily means that the bacteria which cause the disease are present when the chill takes place.

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Nasal catarrh is the commonest chill disease and the one whose pathology can be most easily worked out. A great deal of confusion has arisen in this disease owing to the difference between the chill factor and the bacterial factor, which are here clearly marked, not being recognised.

I have carefully investigated a great many cases of nasal catarrh, and, while some are due to direct infection, which will be considered afterwards, the majority are due to the direct chilling of a cutaneous surface, the first effect of which is a great increase in the secretion of nasal mucus. The bacteria present are those normally found in the nasal and buccal cavities. There does not seem to be any specific bacillus in nasal catarrh, but the bacterial factor is the increased growth of the staphylococci, streptococci, and the varieties of *B. diphtheriæ* present when the chill is contracted, and, unless this growth can take place the symptoms of acute nasal catarrh will not be seen. These bacteria are always present in the mouth and nose, yet they, fortunately, always do not grow there or we would always have catarrh. Some change must take place in their environment before growth can take place; the change induced by the chill factor of the disease allows this, and how this change is caused my experiments show. The result of a large number of incubations of the normal bacteria present in the mouth show:

(1) That the bacteria normally present in the mouth and nose will not increase if incubated in healthy mucus.

(2) That they will grow rapidly in the secretion from the first stage of catarrh.

(3) That the bacteria from a case of nasal catarrh grow slowly or not at all in normal nasal secretion, but very rapidly in catarrhal secretion.

These results show that the normal secretions of the body have an inhibiting effect on the growth of bacteria, but this we knew in many cases before. What we did not appreciate was the most important fact that this power of inhibition can be lost by the reflex effect of chill. If the change in the secretions of the nose which is the first effect of chill in producing nasal catarrh goes far enough to allow of the growth of the bacteria already present, then this growth will produce the symptoms of the disease. If no bacteria were present, catarrh would not go beyond the initial chill effect of increased mucus-secretion.

So from these results in the case of nasal catarrh it can also be deduced that in the secretions of the various cavities in the body which are liable to bacterial invasion the first line of defence exists; that these secretions form a protection from the growth of those bacteria which are always present in the air and our surroundings and whose growth in the secretions is the cause of our chill diseases. This barrier always exists in health and can only be broken down by an alteration in the character of the secretion. This alteration is usually the reflex result of chill or the stimulation of a cutaneous surface, but the same alteration can take place by direct stimulation of a mucous membrane, as seen in some cases of bronchitis and some gastric and intestinal catarrhs.

In all chill diseases the serious symptoms are produced by the growth of bacteria in the secretions of the organ affected; if this growth does not take place the disease rapidly subsides. For instance, in ordinary bronchitis the first effect of chill is the congestion of the bronchial mucous membrane and increased secretion of mucus; if this mucus remains sterile the congestion will soon be recovered from, but if the bacteria present are able to grow in the secretion the bronchial inflammation will increase and spread as the irritation caused by the bacterial toxins in the bronchial secretion is added to the congestion produced by chill.

The most common chill disease is nasal catarrh, the most important pneumonia and rheumatic fever. Between these are the various lung diseases due to chill, such as bronchitis and pleurisy, the pleurisy due to chill being, I think, the pneumococcic variety. Practically all pleurisies are pneumococcic or tubercular. We know that the growth of the pneumococcus can be induced by chill, but we do not know enough about the conditions of growth of the tubercle bacillus to know how tubercular pleurisy commences.

There are also a group of intestinal catarrhs in which, as a result of chill, fermentative diarrhœa occurs owing to the increased growth of the intestinal bacteria, which increase must be due to an alteration in the intestinal secretion. We see these cases sometimes in hot weather in this country, and they are very common in the tropics.

The question of chill after operation is a most important one, especially in abdominal operations.

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We have seen that we have in the natural secretions a protection against bacterial growth, and that this protection is lost by chill from exposure of a cutaneous surface. This emphasises the necessity for prevention of chilling of the body in prolonged abdominal operations. Apart from shock the pneumonia which sometimes follows abdominal operations is due to chill, and chill may also cause the loss of that power of inhibition over the growth of bacteria that the peritoneum possesses. After parturition chill may also cause a change in the vaginal secretion which has these inhibiting properties well marked, and may thus destroy one of the best safeguards against puerperal infection. This is a point overlooked in modern management of confinements.

The bacterium which causes rheumatic fever has not been definitely isolated, but we know from experience that its growth is induced by chill. From this reason, and also from the characteristic that one attack causes susceptibility instead of immunity will, I believe, eventually be demonstrated the fact that the microbe that causes the disease is one of those normally present in the body.

While originally the growth of the bacteria which cause this class of disease is due to chill, continued growth in an altered environment may alter their character and they may become infective. This is often seen in nasal catarrh, more rarely in pneumonia, and there have been cases reported which seem to show that rheumatic fever may sometimes become infective.

One of the great distinctions between chill diseases and infective diseases is, that although both are due to bacterial growth they vary very much as to immunity from further attacks. Infective diseases confer immunity, while chill diseases rather cause increased susceptibility. The reason that this is so is clearly seen from the different conditions under which the bacteria in these divisions grow. The growth of the infectious class is caused by infection of the blood-stream, which acquires the power of immunity from the growth of the bacteria, causing the disease with which it is infected. On the other hand, in the chill diseases the growth of the bacteria commences in the secretions and is due to their alteration by the reflex stimulation of chill; they thus lose their perpetual immunity. This alteration in the character of the secretion when due to a reflex cause is, like all reflexes, more likely to occur in that particular form when once it has begun.

Perhaps the most important thing to be said about treatment is that prevention is better than cure. In youth the body should be accustomed to a hardening process by cold baths and careful exposure, so as to make the body as far as possible what the thin-clothed lascar meant when he was asked why he did not seem to feel the English cold weather, "My body all face."

August 6th, 1906.

THE therapeutic value of casein combined with glycero-phosphate of sodium has received a very large measure of clinical recognition, and lately the successful results obtained in the treatment of certain serious types of anæmia in which iron proved ineffectual have drawn attention to the use of sanatogen, which consists of 95 per cent. casein and 5 per cent. glycero-phosphate of sodium. In the difficult position of deciding the diet for marasmic children medical men now find a ready solution of the problem by ordering half a teaspoonful of sanatogen to be mixed into a paste with water and given in milk. In addition to the clinical evidence which clearly indicates the valuable assistance to be gained by the administration of sanatogen it is not only noteworthy, but also of scientific interest, to observe that by the refractometric estimation of proteids as a control of therapeutic feeding the advantages derived from this preparation can be accurately estimated. This method has been utilised to determine the effect of sanatogen in a series of patients suffering from the most various complaints, and the conclusion arrived at by the investigators was that the combination of casein and glycero-phosphate of sodium had a very beneficial effect. This favourable opinion was formed after the most laborious and painstaking observations on the proteid contents of the blood-serum of the patients. So carefully were these investigations carried out that, in order to determine accurately the degree of possible variations in the proteid contents of the blood-serum and to exclude other influences, the refraction point of the blood-serum was estimated for a period of eight to fourteen days, according to the nature of the case before the sanatogen diet was taken. It is undoubtedly most satisfactory to be able to confirm the teaching of clinical experience by the accurate findings of experimental research.

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WHAT ARE THE DISEASES OF THE STOMACH FOR WHICH SURGICAL INTERFERENCE MAY BE BENEFICIAL?*

By W. HALE WHITE, M.D., F.R.C.P
Physician to Guy's Hospital.

GENTLEMEN,—Perhaps the greatest change that has taken place in the practice of medicine during the last thirty years is that our responsibility has enormously increased. Formerly in many instances all that we could do was to keep the patient quiet in bed and watch him, often until he died; now we have to quickly decide whether we cannot by calling in the aid of surgery, save his life. In no instances is this change more marked than in those in which the stomach is the seat of disease, and therefore I hope it will not be altogether profitless if this evening we discuss the conditions in which surgery may aid those suffering from disease of that organ.

Perforation of a gastric ulcer into the general peritoneal cavity is always or almost always fatal if the abdomen is not opened, and therefore we will consider this first. It is not an uncommon event; last year ten cases were admitted into Guy's Hospital, four were saved; thirty years ago all ten would have died. Clearly, therefore, it is of the greatest importance to recognise the condition. For practical purposes there are two groups of diseases with which a perforated gastric ulcer may be confounded; the first is comprised of those for which a laparotomy is not needed and is, therefore, if possible, most certainly to be avoided; the second is comprised of those for which a laparotomy is necessary, and here a mistake is not so serious. In the first group we have two conditions which in this country we may neglect, namely cholera and dysentery, but I have known ulcerative colitis give rise to sudden severe pain very like that of a perforated gastric ulcer; this, however

* Delivered before the Ilford Medical Society.

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is very rare. Let us never forget lead colic, renal colic, and biliary colic; they have been mistaken for a perforated gastric ulcer, but they do not produce signs of peritonitis nor a rapidly mounting pulse. Most cases of acute pancreatitis are operated upon under the impression that they are instances of perforated gastric ulcer; for in both there is sudden severe abdominal pain at the upper part of the abdomen, with tenderness, and the patients are rapidly collapsed; but although it is true that with care a successful diagnosis of acute pancreatitis may be made, the chances of such a diagnosis being right are not sufficiently great to withhold from the patient the benefit that will probably follow laparotomy if the diagnosis is wrong, for in that case he is either suffering from a perforated gastric ulcer or intestinal obstruction, and in both of these his only chance lies in a laparotomy. We must always remember that the first evidence of acute pneumonia may be severe pain in the abdomen. I am sure it is not an uncommon mistake to believe that a patient with acute pneumonia has a perforated gastric ulcer or intestinal obstruction; perhaps it is those cases in which the diaphragmatic surface of the lung is affected that most resemble intra-abdominal disease, for in these cases the abdominal muscles may be rigid, possibly owing to the great pain that would follow movement of the diaphragm. The best guides are the rapidity of respiration, the initial rigor, and the physical signs in the lungs. We must all strive hard to avoid this mistake, for the chances of death are certainly increased if we open the abdomen of a patient with acute pneumonia. I quite acknowledge that very, very rarely hysteria may give rise to difficulty, but we should show the utmost caution in diagnosing hysteria, for such a diagnosis, if wrong, may cost the patient her life.

We now turn to the second group of conditions which are liable to be confounded with perforation of a gastric ulcer. Acute strangulation of the bowels, very severe appendicitis, and acute peritonitis, usually due to rupture of an abscess in connection with the appendix or to sloughing of the appendix, are the commonest members of this group, but others are a twisted ovarian tumour, a ruptured extra-uterine pregnancy or pneumococcal peritonitis. We should try our utmost to separate these from a perforated gastric ulcer and from each other, but a mistake here is less serious than in the first group, for laparotomy is, in the present state of our knowledge, the right treatment for all members of the second group.

Let us suppose that we have decided that a gastric ulcer has perforated into the peritoneal cavity. There is no doubt as to what we shall do next—namely get someone familiar with abdominal surgery to open the abdomen at once and sew up the ulcer. Every hour that this is delayed seriously increases the chances of death. Mr. Mayo Robson gives the statistics of many cases and finds that among those operated on under twelve hours from the time of perforation the mortality is 28·5 per cent, among those operated upon in from twelve to twenty-four hours it is 53·6 per cent., among those operated on in from twenty-four to thirty-six hours 87·5 per cent., and among those operated upon in from thirty-six to forty-eight hours 100 per cent. These figures speak for themselves, and show how great is the responsibility we bear, and how necessary it is to promptly come to a correct diagnosis. Occasionally, but very rarely, the perforation takes place into some adhesions previously formed around the ulcer, and then a subphrenic abscess results. This must, of course, be treated surgically. Therefore, whenever perforation takes place the case is one for surgery and if we fail to diagnose it and to employ surgery the patient will die, but if we treat it correctly there is fair chance that surgery will lead to recovery.

Let us next consider some of the chronic effects of gastric ulcer. One of these is dilatation of the stomach. This may take place either because an ulcer near the pylorus by its cicatrisation partially occludes the pylorus or because the ulcer, by adhesions it forms to neighbouring structures, kinks the pyloric end of the stomach, and so obstructs the exit of food. Two points are worthy of mention: first chronic dilatation of the stomach not due to an organic cause rarely reaches an extreme degree, secondly the vigour of peristalsis of the gastric wall as seen through the abdominal wall is evidence that the dilatation is due to organic obstruction. A man was in the hospital under me a few months ago; he frequently vomited three quarts at one gush, and gastric peristalsis was very striking. There were strong reasons for believing that he was not suffering from a gastric carcinoma, and, therefore, although

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there was absolutely no history of gastric ulcer, yet as the degree of dilatation and extreme peristalsis pointed very strongly to an organic obstruction at the pylorus, a cicatrising gastric ulcer was diagnosed. Sir Alfred Fripp opened the abdomen, the diagnosis was confirmed, a gastro-jejunostomy was done, and since that the patient's health has improved vastly. I could quote other instances, and would lay it down as a proposition that when a gastric ulcer leads to much dilatation of the stomach a short-circuiting operation should be performed; indeed, it is difficult to see what else could be done. Drugs certainly cannot overcome the obstruction, and the benefit that often follows a gastro-jejunostomy is very striking, the patient becoming really quite another man; but inasmuch as operation for dilatation of the stomach not due to pyloric obstruction does so little good as not to be justifiable in certainly the great majority of cases, much responsibility rests with us, and we must employ our utmost powers to determine whether or not the dilatation is due to organic obstruction.

Gastric ulcers have other effects on the stomach besides leading to its dilatation. In the post-mortem room we find that in 45 per cent. of all the cases of gastric ulcer some local peritonitis outside the ulcer has led to the formation of adhesions between the stomach and most commonly the pancreas, but very often the liver and less frequently the colon.

Probably these adhesions generally give rise to no symptoms, but often they may cause pain, usually at the upper part of the abdomen; and sometimes at least patients who are unjustly set down as neurotic dyspeptics or sufferers from gastralgia or gastrodynia really owe their troubles to these adhesions. It is a curious fact that the pain is often paroxysmal; perhaps this is due to dragging on the adhesions by contraction of the stomach. These paroxysms may be very severe; I have seen them cause a patient to writhe on the floor and induce a well-meaning man to send for his doctor in the middle of the night. They may occur at frequent intervals for many years—I have known them go on for thirteen. Often there is more or less constant pain as well; indeed, constant pain is sometimes the chief feature, but it is important to remember that the pain may be only paroxysmal; there are no long intervals of freedom from pain as is often the case in ordinary dyspepsia and in that due to an actual ulcer. If the ulcer has healed and all the pain is due to adhesions, it is not made worse by food, the patient is not wasted, the tongue is clean, there is no vomiting, and there may be no dyspepsia. Sometimes tenderness is complained of over the adhesions; often it can only be elicited by deep pressure; probably then it is due to our actually dragging on the adhesions by our pressure. If the adhesions are numerous they may form a mass that can be felt through the abdominal wall, giving an ill-defined sensation of matting. It might be thought it would be difficult to diagnose these cases; but, fortunately, we frequently have a history of gastric ulcer to help us, and even when there is not it is often possible to arrive at a correct conclusion; the whole pain seems so real that it is difficult not to ascribe it to organic disease. When once we have arrived at this diagnosis it is clear that the use of drugs should cease, for the only drug that would stop the pain is morphia, and if this be given the patient may become a morphia-taker. The sufferer has two choices: either he can put up with his pain or a surgeon can be asked to open the abdomen. Probably when the adhesions tightly bind the stomach to some neighbouring organ severe pain will not be so striking as when the adhesions are of some little length; and it, fortunately, is just these it is most easy to deal with. They should be cut entirely away, not simply divided; for if this is done they will re-form, and indeed they may re-form even when taken entirely away. If the patient is a woman and the symptoms have lasted a long while, there are often many subjective symptoms added to the pain due to the adhesions; these may remain after the adhesions are taken away; indeed they may be so prominent that it is unwise to operate, just as often it is unwise to fix a floating kidney, for no remission of the subjective symptoms follows. Sometimes adhesions to the stomach are caused by disease outside of it; for example, a mass of adhesions due to inflammation around the gall-bladder induced by gall-stones may press upon the pyloric end of the stomach, but more frequently it is the duodenum which is involved. Such adhesions may cause dilatation of the stomach or paroxysms of pain. These perhaps hardly come in the scope of this paper, but I am sure it is often forgotten that a large mass of

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thick peritoneal adhesions may form as a result of gall-stones even when the gall-stones do not ulcerate out of the gall-bladder.

Let us now leave this part of our subject and turn to gastric hæmorrhage. No one would think of asking a surgeon to interfere for hæmatemesis due to cirrhosis or cancer, therefore we are narrowed down to cases in which a gastric ulcer is diagnosed. My first contention will be that this is often thought to be present when it is not. It is allowed that the malady commonly diagnosed as gastric ulcer is very much more frequent in women than in men; also that many more cases in women occur in the two decades from twenty to forty than in any other two decades, while in men the disease is commoner later in life. Further, many collections of cases show that the actual mortality in men is about the same or even a little higher than in women. Because of these facts one of two conclusions must follow: either the outlook of gastric ulcer in women is much more favourable than in men, or many cases occurring in women and classified as gastric ulcer at the bedside are really instances of some other malady. I have long thought that the last is the correct alternative and I shall now prove my point. Last October there was admitted into Guy's Hospital under me a woman just over fifty years old. She had had dyspepsia and hæmatemesis for nine years and she was admitted for severe hæmatemesis. On account of her age she was thought to have a genuine gastric ulcer, and it was decided to try to stop the hæmorrhage by medical means and then when she had recovered from the loss of blood to operate with a view of doing a gastro-enterostomy. She died from her hæmorrhage. At the post mortem not a trace of an ulcer could be found. In the 'Lancet' for June 29th, 1901, I published an account of the case of a woman who had had gastric pain, vomiting, and hæmatemesis for seven years. She died from hæmatemesis. At the post-mortem examination no ulcer or source of the blood could be found. And I also mentioned the case of a woman who had been attending out-patients for years for gastric pain, vomiting, and hæmatemesis; she was taken into the hospital, the stomach was opened; no source for the bleeding was discovered. I was able to give others taken from medical literature, and quite recently Dr. Bertrand Dawson ('Brit. Med. Journal,' Oct. 21st, 1905) has published several cases—for example, a female æt. 25 years. For two years she had had pain, vomiting, and hæmatemesis. She was anæmic; there was no wasting. First attack, August, 1900, in bed two months; second attack, February, 1901, in bed six weeks; third attack, January, 1902, fed on nutrient enemeta; fourth attack, July, 1904. Operated on during fourth attack, a small erosion on the posterior wall of the stomach which oozed blood, base ligatured. Many fresh hæmorrhages after operation; patient's condition not improved. A second operation was performed for another attack of hæmatemesis, nevertheless the hæmorrhage has continued. Another case was a woman, æt. 28 years, who had had pain, tenderness, and vomiting and hæmatemesis for eleven years; at operation nothing was found but a congested patch of mucous membrane. A third case, a woman æt. 30 years, had had pain, vomiting, and hæmatemesis on and off for twelve years; at the operation there was only a small abrasion from which blood was oozing; the pain and vomiting continued after the operation.

I have now given you enough cases to show that there is a disease closely resembling gastric ulcer, for its main symptoms are gastric pain, vomiting, hæmatemesis, and tenderness over the stomach, but no ulcer is present. Occasionally but by no means always the surgeon when he opens the stomach sees a slight abrasion of the gastric mucous membrane from which the blood is oozing but it must be remembered that the blood could not come through the epithelial and submucous coat unless there was an abrasion. Sometimes there is a general oozing from the gastric mucous membrane, sometimes the oozing occurs in small or large patches. We need much more information about these cases, but as far as I know the disease occurs almost entirely in women, not in children; the patients are often not wasted as much as the prolonged history of severe vomiting and dyspepsia would lead us to expect; we never meet with any of the mechanical effects of ulcer, such as perforation, dilatation, or adhesions; and the pain probably does not bear the same relation to food as in genuine ulcer; often amenorrhœa is present and it is said that the patients may be chlorotic, but we want more exact information on this point. The reason why I insist on these cases here is that we certainly should not operate for the hæmate-

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mesis which follows from them. One case I have just mentioned showed that the bleeding went on after operation, and you can hardly expect surgery to deal with an oozing. Mayo Robson, speaking of these cases ('Brit. Med. Journ.,' Sept. 30th, 1905), says operation is seldom required and rarely justifiable, and in the 'Lancet' for March 9th, 1901, he refers to five cases which died. The right treatment for these cases, I believe, is to put them to bed, give them plenty of good nourishing food—it need not be slops or fluid diet—and to give iron regularly. I have seen these patients improve rapidly on this treatment. If the bleeding is serious you may give adrenalin, or large doses of the Liquor ferri perchloridi, say a drachm at a time, with an equal amount of glycerine.

Having put these cases aside, we now come to those in which there is hæmatemesis from an actual ulcer. The first point I would mention here is that, if possible, operation should not be done during an attack of hæmorrhage, for it is rarely directly fatal, and when the patient is very anæmic he is not in a good condition for a long operation and the healing of his gastro-enterostomy. The guide to operation for hæmorrhage is not so much the severity of the bleeding as its frequent recurrence. There are many patients whose health is undermined by recurrent hæmorrhages from gastric ulcer; they should certainly be handed over to the surgeon and a gastro-enterostomy will often be quite sufficient to lead to healing of the ulcer with cessation of the bleeding. It is rarely that the bleeding point itself can be dealt with; it may be in a dense fibrous ulcer adherent to the back of the abdomen.

Talking of these cases of repeated bleeding brings me to those not uncommon examples of long-continued suffering from the pain, dyspepsia, and vomiting of gastric ulcer. Such patients may be thin and wasted, afraid to eat, weak and always in miserable health. For such as these medicine can do little; so long as food is constantly put into the stomach the ulcer hardly has an opportunity of healing. The benefit that can be conferred by surgery is very striking. Take the case of a man whom I saw: he was thirty years old, quite unable to follow his occupation as an engineer, and therefore unable to marry his betrothed. From 1894 to 1897 he was treated with bismuth and a restricted diet. In December, 1897, he suffered from sickness, hæmatemesis, and pain. He was kept in bed some time. In the spring of 1899 he lost ground; during the winter of 1899–1900 he was in continuous pain; in the spring of 1900 he had much hæmatemesis and severe pain; he lost 20 lb. in weight. What can drugs do in a case like this? Little or nothing. But we handed this patient over to a surgeon. He was operated upon, gained weight and strength directly after, married five months later, and a year after the operation was in excellent health and had not been away from his work for a day since his marriage, although before operation he was very wasted and so weak that he could not walk. Precisely what operation should be performed will depend upon what is revealed when the stomach is opened, but it will nearly always be found that a gastro-enterostomy is the best thing to do; the food is then diverted from the ulcer and so it has the opportunity of healing. It must not be thought that no cases of gastric ulcer will get well without surgery; many get perfectly well by the usual medical treatment, but surgery will often lead to a cure in those patients whom ordinary medical means have failed to relieve.

Tetany is a serious complication of gastric ulcer; it is usually associated with dilatation; and, inasmuch as it is often fatal, and gastro-jejunostomy will relieve the dilatation, it is clear that this operation should be done if tetany occurs as a complication of gastric ulcer.

Lastly, we come to carcinoma of the stomach. Cases have been recorded in which the growth has been excised, but it must be very rarely that this can be done with success; indeed, I myself have never seen one. Possibly this is because all the patients who come under me are either so ill that a second opinion is sought, or so ill that they come into a hospital. By the time I have come across these patients the growth is either so large or so adherent to neighbouring structures that excision is impossible, or there are so many secondary growths that removal of the gastric tumour is inadvisable. The most promising case that has come under my care was that of a man from whom last spring Mr. L. A. Dunn removed a carcinoma of the pylorus and several glands which were secondarily affected; after the operation the patient improved very much and gained considerably in weight, but in the autumn he was

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re-admitted into the hospital with secondary growths in various parts of his body. The diagnosis of gastric carcinoma will have to be reached much earlier than at present if we are to excise the growth successfully; indeed, some have urged that the abdomen should be opened and the stomach inspected in any case of doubt. Although the removal of the growth is very unpromising, yet often surgery can afford great relief to the miserable sufferers from this disease; for frequently the growth obstructs the pylorus, and then a gastro-jejunostomy may be of much service, for the food no longer causes pain by passing over the growth and it goes straight into the intestine instead of remaining to decompose in the stomach. It must, however, be put clearly before the patient that such an operation is only palliative. It is our duty to tell these patients that although the growth cannot be taken away operation may, if successful, prolong life a few months and save suffering; it will then be for the patient to decide, but if he is adverse you can hardly urge him strongly to have an operation performed, especially as there is always the risk that the operation itself may prove fatal. Not long ago Mr. Fagge did a gastro-jejunostomy for a patient of mine in whom the carcinoma of the stomach was so extensive that there was only just room to do the short circuiting; yet he was delighted at the result, for he gained weight, his appetite returned, his vomiting stopped, and, I think, the suffering of his last days is less than it would have been had the operation not been performed.

The last condition to be mentioned is gastroptosis. I doubt whether any of us here present have sufficient experience of operation for this to say much about it, but my impression is it will not be found to be of much use.

August 13th, 1906.

MEDICAL men sending patients to the "Cornish Riviera" will note with satisfaction that the Great Western Railway have now much improved the travelling facilities to the West of England. The "Cornish Riviera, Limited, Express" leaves Paddington station at 10.30 in the morning and reaches Penzance at 5.10 in the afternoon. This train does not stop till it arrives at Plymouth, and is provided with luncheon cars, so that the passengers are well cared for in every way. This information is from a pamphlet on the new direct route to the Cornish Riviera, sent to us by Mr. J. Morris, Superintendent of the line, G.W.R., Paddington.

A LECTURE
ON
PROSTATECTOMY, ITS PREVIOUS AND AFTER TREATMENT.

By H. L. BARNARD, M.S., F.R.C.S.,
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GENTLEMEN,—In this lecture I shall refer only to suprapubic enucleation of the whole prostate with the finger, and I do not propose to enter into elaborate details of the method of performing this operation. There are, however, several practical points about this matter which contribute in no small degree to the success of the operation, and it is with these that I propose to deal.

THE PREVIOUS MANAGEMENT OF THE CASE.

One of the first questions which naturally presents itself is, At what stage of prostatic enlargement should the surgeon recommend the patient to have his prostate removed?

It is certain that the condition is a progressive one, and that the longer the prostate is left to enlarge the greater will be the obstruction to the passage of urine, and the more will the kidneys and bladder be damaged by this obstruction.

The only alternative treatment in cases in which there is considerable obstruction is the use of a catheter once or twice a day. This catheter is passed with a double object. In the first place, it draws off a large proportion of the urine, which has not therefore to be forced through the obstructed urethra. The amount of straining of the bladder-muscle is therefore reduced to a minimum, and the sacculation of the bladder and dilatation of the ureters and kidney which this straining produces progress as slowly as possible to their fatal termination. In the second place, the catheter empties off twice a day the residual urine in which micro-organisms are probably thriving and in this way keeps under, although it cannot be said to cure, the cystitis. On the other hand, if the patient is careless or through poverty cannot afford the many contrivances which render catheter life safe and convenient, he probably counterbalances the good he does through completely emptying his bladder by the frequent re-infections he introduces during the process.

It might, then, be said that the prostate should be

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removed as soon as it has produced any evidence of obstruction, and no doubt this is theoretically correct, and I believe we shall attain to this standard some day. It is a recognised law of surgery that when even an innocent and slow-growing tumour presses on some structure which is essential to life that tumour should be removed, and the sooner the better. Acting on this rule, thyroid adenomata are removed at the first sign of pressure on the trachea or œsophagus; laminectomy is performed for tumours of the spinal membranes when the first signs of paraplegia appear, and tumours pressing on the intestines are similarly attacked. From a similar reasoning it would seem right to remove a prostate as soon as there is any sign of obstruction to the flow of urine. In practice, however, it is at present better to wait and prescribe catheter life for a time. When the patient has had one or perhaps two attacks of retention, and certainly after the first attack of septic cystitis, then the surgeon should strongly recommend prostatectomy, for the bladder when once infected can never be entirely freed from micro-organisms until the obstruction is removed and it is able to empty itself frequently and entirely. Even where no infection or retention has occurred a greater quantity than three or four ounces of residual urine would indicate that the prostate should be removed at once.

There are two objections to the performance of prostatectomy at the earliest stage of obstruction. The first is that the public are not yet educated up to it and cannot appreciate that so serious an operation is advisable when their symptoms are so slight. After an attack or two of retention and cystitis and some months of catheter life they are prepared to accept any reasonable risk to escape from their present discomfort and their future fate.

Other good reasons for postponing prostatectomy are that large prostates are more easily removed than small ones. The hæmorrhage is, as a rule, less severe, and there is less risk of stricture of the neck of the bladder after the operation, although this complication very seldom occurs.

The indications for prostatectomy, then, are: (1) when catheter life has become established in consequence of one or two attacks of retention; (2) when chronic cystitis with acute exacerbations has commenced; (3) when a vesical calculus is present; (4) when the prostate is large, soft, and elastic, and associated with three or four ounces of retained urine; (5) a medium-sized prostate with cystitis or calculus.

Small fibrous contracted prostates are very difficult to deal with by prostatectomy; they must be torn out in fragments with forceps or broken up and cut away with curved scissors. I have only operated on one such case and he gave a better result than any other. He gained 2 st. in weight, lost his bronchitis, and went back to the work he had abandoned in consequence of his urinary trouble.

The contra-indications to prostatectomy are: (1) very great age where catheter life is giving fair results, but the matter of age is relative; (2) in younger patients where catheter life is easily borne and there is no cystitis or stone; (3) in those whose condition borders on uræmia, as indicated by rapid wasting, urinous breath, a very low specific gravity to the urine, and great feebleness.

Loss of expulsive power in the bladder is no contra-indication to prostatectomy, for when the obstruction is removed this will often return in a remarkable degree. Even a double hernia should not deter the surgeon from this operation, for hernia is, in most cases, a consequence of the urinary obstruction, and will be more easily retained by a truss when the obstruction is removed.

The bronchitis which is often present in cases of prostatic hypertrophy would indicate the use of chloroform as an anæsthetic rather than of ether. I believe that this bronchitis is often a consequence of the urinary trouble, and is due to the absorption and excretion by the lungs of the products of decomposition of urine in the bladder, notably of ammonium carbonate. I have certainly seen bronchitis clear up in a most surprising fashion after the prostate has been removed.

The preparation of the patient for operation.—When it has once been settled that prostatectomy shall be performed, the next thing is to prepare the patient, so that he may pass through the operation successfully, and with as little risk as possible.

The operation must never be attempted during an acute attack of retention or cystitis. These must first be subdued by the regular use of the catheter, by the prescription of urinary antiseptics, and perhaps by vesical lavage. The operation must only be undertaken when all acute symptoms have subsided.

The most important thing, however, is to render

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the patient's urine as little septic and irritating as possible, for the raw, ragged cavity left by the removal of the prostate must heal in a pond of such urine as the patient possesses. When urine is alkaline it deposits phosphates on raw surfaces. It is therefore of the greatest importance to render the urine acid before operation. This can be achieved in nearly every case by the use of two drugs—acid sodium phosphate and urotropin well diluted in water. Acid sodium phosphate (NaH_2PO_4) is the salt which gives to urine its normal acidity. Dr. Robert Hutchison was the first to suggest its use to render the urine acid. The drug has no poisonous effects, but when given in excess it produces diarrhœa, which, however, is not a bad thing in prostatic cases, so long as it does not exhaust the patient.

The form in which I usually prescribe these drugs is as follows:

Acid sodium phosphate . .	1 drm.
Urotropin	5 gr.
Water	1 pint.

The patient drinks three or four pints of this beverage per day.

This is known as the "acid flood" to distinguish it from the "alkaline flood," in which a drachm of potassium citrate is given in a similar manner to render the urine alkaline in certain forms of acid cystitis due to the *Bacillus coli*.

A word of warning must be given about the exhibition of urotropin in urinary cases with damaged kidneys. It is liable, even in small doses (5 gr. three times a day), to produce hæmorrhagic nephritis if its use is long continued. This warning is all the more necessary because the presence of blood in the urine is not a matter of surprise in the cases in which urotropin is prescribed. By careful observation, however, the cases of urotropin poisoning may be distinguished. The two signs on which I rely are that the condition of the urine is largely one of hæmoglobinuria, and that the temperature rises steadily with the appearance and increase of the blood during three or four days, until it reaches a height of 102° or 103° F. The presence of hæmoglobin is recognised by the brownish colour of the urine. When the sediment has settled the supernatant fluid, although still of a deep colour, is transparent and clear. And when a fresh specimen of this porter-coloured urine is examined under the microscope not nearly enough red corpuscles are seen to account for its deep colour.

I have known a prostatic case take 5 gr. of urotropin three times a day for two months before symptoms of renal irritation appeared. Directly the urotropin was discontinued the temperature and blood declined and disappeared in three or four days.

The use of the "acid flood" will render a stinking ammoniacal urine acid and sweet in three or four days in nearly all prostatic cases. I cannot speak too highly of this combination, which should entirely replace the feeble mixture of ammonium benzoate and boric acid which formerly was the best at our command.

In rendering the urine acid we shall to a large extent have freed it from micro-organisms. The most practical division of urinary micro-organisms is into those that live in alkaline and those that can grow in an acid urine. The former are more common and produce severer forms of cystitis. If we succeed in changing the urine from an alkaline to an acid medium these micro-organisms cannot grow and die out. They are, moreover, very sensitive to the action of urotropin, and no doubt the change of reaction is largely due to the death of those micro-organisms which convert urea into ammonium carbonate. But, unfortunately, at least some varieties of *B. coli communis* can flourish in acid urine, and this micro-organism is not affected by urotropin. So that it may come about that even after we have rendered our prostatic patient's urine acid he may still continue to secrete pus, and the microscope may show that his urine is swarming with the colon bacillus. In one or two cases I have been successful in removing these by turning the urine alkaline for a week or so with potassium citrate and the "alkaline flood." It has always puzzled me why this should act when the same colon bacilli were living only a week or two before in alkaline ammoniacal urine. I can only suggest that the "alkaline flood" acts mechanically by throwing down a fine precipitate of the earthy phosphates, and that the actively motile bacilli stick to these minute particles and are removed mechanically with them. However that may be, it is often useful to change the reaction of the urine once or twice, and in this way it may be finally rendered acid and practically free from

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micro-organisms, and this is the condition into which it should be brought before prostatectomy is undertaken.

It will not be necessary to wash out the bladder in the great majority of cases, but should the means to which we have already referred fail, or act but slowly, then vesical lavage may be resorted to. The best solution to employ is one of oxycyanide of mercury in the proportion of 1 in 2000. It may be used as strong as 1 in 1000 without ill-effect, and in the urinary clinics of Berlin I constantly saw it employed in this strength, but personally, I always employ the more dilute solution. This drug is a powerful antiseptic, does not irritate the bladder, and produces no poisonous symptoms. It is little known in England and there is some difficulty in getting it, and I must warn you not to employ the cyanide of mercury in its place in the same concentration of solution.

A still further preparation for operation has been suggested by Professor Wright. Where one must operate in the presence of the *Bacillus coli communis* Professor Wright has suggested that by the injection of dead cultures of this micro-organism the immunity of the patient may be raised in three or four days, even to as great an extent as five times. When during the operation the micro-organisms are rubbed into the raw tissues those tissues will be five times more resistant to infection than if no such vaccination had taken place. I have not had an opportunity of trying this method as yet, but it appears to be based on sound principles.

Practical Points about the Operation.

I do not propose to describe the operation in detail. Shortly it is performed as follows: The bladder is opened supra-pubically. One or two incisions are made on the prominent parts of the prostate. The index finger tip is then worked into these cuts and enucleates the entire prostate. Counter-pressure is made by the first and second fingers of the other hand inserted into the rectum. If the prostate is removed in one piece the prostatic urethra is sacrificed. If the lateral lobes are removed separately through two incisions the urethra can be spared.

Now as to practical points.

It is well to employ the Trendelenberg position for this operation. The hæmorrhage is for the most part venous, from the prostatic plexus, and raising the pelvis very considerably reduces it.

When the bladder has been opened and a finger is inserted to examine the prostate the post-prostatic pouch should also be investigated for a stone. This pouch is often deep, and unless specially looked for, a stone may easily escape observation. Should it do so, the suprapubic wound will probably fail to close, and cystitis will persist after the operation and resist all the efforts of lavage to cure it. Finally, the surgeon will have to open up the bladder again and remove the stone. This mistake is only likely to occur where the surgeon is endeavouring to perform the operation in a phenomenally short time.

One of the great difficulties about suprapubic enucleation is that the fingers of one hand are in the rectum for counter-pressure, and should not, therefore, be inserted into the bladder immediately after, yet it is exceedingly difficult to cut on the prostate with curved scissors or a knife without the index finger of the other hand to guide the incision. This difficulty has been overcome in all sorts of manners. One surgeon uses a rubber glove for the rectal hand, and has it removed when he wishes to use that hand for the bladder. Another employs an assistant to push up the prostate whilst he incises the floor of the bladder and only inserts his own fingers in the rectum when he commences to enucleate. The latter parts of the operation he performs with one hand. One surgeon is reputed to grow his index finger nail long and harden it in alcohol, and then sharpen it to a point with which he scratches through the floor of the bladder. In this way he dispenses with scissors and knife.

Most people, while appreciating the convenience of this procedure, will be prejudiced against the method itself, and I have therefore modified the adenoid curette (see Fig.) so that it terminates in a blunt knife edge. I have employed this instrument in two cases, and it has been perfectly successful and very convenient. By its use the whole enucleation of the prostate can be carried out with only one index finger in the bladder.

The hæmorrhage in prostatectomy is not often severe, especially if the Trendelenberg position be employed; but should it be so, hot water and adrenalin chloride will usually control it with sponge pressure. The hot water, which must be very hot, is best irrigated into the cavity left by

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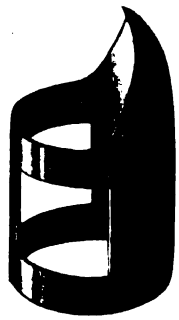
the prostate by a catheter passed down the urethra. Adrenalin chloride, which is less efficient because the hæmorrhage is usually venous, is applied on a good-sized sponge in a holder thrust into the prostatic cavity and held there for a minute or so.

The whole success of a case of prostatectomy turns on securing perfect drainage. Urine is a non-particulate fluid capable of infiltrating cellular tissues widely and under very slight pressure. It is not like blood and pus, which have apparently been evolved as particulate fluids to prevent their wide diffusion and extravasation. For blood and pus when they escape into the tissues block up the minute channels with their particles and hinder further escape.

The application of this principle to prostatectomy



is perfect drainage. I usually insert two large tubes down to the floor of the bladder and one into the cavum Retzii. Some surgeons tie in a catheter in addition, but I have not found this necessary.

No attempt should be made to suture the bladder or close the abdominal wound.

For seven days the most perfectly free escape of urine must be secured. The open lymph-spaces in the wound will have become closed by inflammatory hyperplasia about the fourth day, and the risk of extravasation will be passed.

The After-Treatment.

Where there is considerable shock after the operation the patient should be stimulated in the usual way with the continuous subcutaneous injection of four or five pints of saline solution and the free use of strychnine and caffeine. The elevation of the foot of the bed will not only lessen venous hæmorrhage but assist in diminishing the shock.

The drainage of the bladder must be maintained absolutely free, and for this purpose the tubes should be irrigated through with boracic lotion night and morning to remove all clots. The irrigation is best carried out with a funnel and tube to which a soft rubber catheter is attached which can be insinuated to the bottom of each tube in the bladder in turn.

Finally, a catheter can be passed *per urethram*, and the ragged cavity left by the prostate well washed out through the suprapubic tubes.

During the whole of the after-treatment it is essential to keep the urine acid and free from ammoniacal decomposition. Otherwise phosphates are deposited in the wound and healing is delayed while these patches of tissue slough away and separate. The irrigation to which we have referred is partly directed to this end, but the most powerful means of achieving acidity is to continue the administration of the "acid flood" composed of the acid sodium phosphate and urotropin.

After seven days efforts must be directed to getting the supra-pubic wound soundly healed and avoiding any stricture at the neck of the bladder. It is, I believe, denied by some surgeons eminent in urinary matters that stricture of the neck of the bladder ever occurs. I have, however, now heard of three cases privately, and there is no doubt that unless special precautions are taken this lamentable consequence may follow prostatectomy in certain cases.

When the prostate is enucleated in one piece the prostatic urethra is of necessity taken away, and even a part of the membranous urethra may be torn out with it. In this specimen you will see that about half an inch of membranous urethra was removed in addition to the whole of the prostatic urethra, and in this case the vesical end of the urethra attempted to granulate over and close up. The condition was, however, discovered in time, and a steel bougie was passed through into the bladder and a catheter tied in. Afterwards a catheter was passed daily until healing was complete. The membranous urethra is the last adhesion, as a rule, in enucleating the prostate in one piece, and it would appear better to cut this with curved scissors and leave as much behind as possible rather than to tear it through, when it may break at any point. Where the prostate is removed in two or more pieces the whole of the prostatic urethra can be preserved, and the risk of stricture is altogether avoided.

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The urinary passage may also become closed at the floor of the bladder, where the sphincter vesicæ is situated. Where the prostate is large, and big adenomatous masses have projected up through the sphincter into the cavity of the bladder there is very little risk of closure, but even then a diaphragm-like projection is often seen in this situation. Where the prostate is small and the sphincter has not atrophied from pressure the risk is proportionately great.

In the case of the patient whose prostate I have already shown you, with part of the membranous urethra attached, the floor of the bladder also attempted to close over. When I dilated his suprapubic wound three weeks after the operation, and passed my index finger into the bladder in order to guide a steel bougie safely through the obstruction, I found the floor of the bladder complete, and the urethral orifice only represented by a dimple. I dilated this orifice freely with my finger so that the raw prostatic cavity freely communicated with the vesical cavity, and then tied in a catheter, which passed right through into the bladder. The man did very well after this, and his suprapubic wound rapidly closed.

The great point is to get the suprapubic wound to close quickly after the seventh day, whilst the natural passage for the urine is kept open by the passage of a catheter daily. The torn end of the membranous urethra will then be opened up daily, and the discharge of urine will prevent the neck of the bladder from closing. Nature, in some mysterious way, appears to be able to keep open an orifice, which is essential. This is very well illustrated in gastro-enterostomy, for if the pylorus is patent the artificial aperture will soon close, but if the pylorus is permanently stenosed the gastro-enterostomy aperture remains open.

We can hasten the closure of the suprapubic wound by removing all tubes from it as soon as it is safe and by sitting the patient up about the seventh day. As long as the patient lies on his back the urine tends to run from the suprapubic wound, but when he sits up the neck of his bladder becomes dependent, and the bladder will hold water and tend to discharge it by the urethra.

A patient cannot be expected to pass water naturally while his suprapubic wound is freely open. This should close sufficiently for natural micturition between the fourteenth and twenty-first day after operation. If the patient has not passed water naturally by the twenty-first day something is probably wrong, and the suprapubic wound should be dilated up and the neck of the bladder explored by the finger and a bougie or catheter passed by the urethra.

Natural micturition may be induced a day or two earlier than it otherwise would by the following device. If a catheter large enough to block the suprapubic wound be passed into the bladder and connected with a funnel, the bladder can be gently distended with boric acid solution until the patient experiences a desire to micturate, which he is often able to do naturally into a bottle. The process can be repeated several times in succession, and not only greatly encourages the patient but most effectually washes out the bladder.

The methods of avoiding stricture after prostatectomy may be summarised as follows:

(1) The prostate may be removed in two or more pieces and the prostatic urethra preserved as far as possible.

(2) Where the prostate is enucleated in one piece and the prostatic urethra is necessarily sacrificed, care should be taken not to tear away the membranous urethra as well.

(3) A catheter may be tied in for twenty-four hours after the operation.

(4) A catheter should be passed into the bladder once or twice a day and free irrigation established out of the suprapubic wound.

(5) The suprapubic wound should be induced to close as soon as possible by removing the tubes from the bladder on the seventh day and sitting the patient up.

(6) Natural micturition should be encouraged as early as possible by distending the bladder gently by the suprapubic wound about the fourteenth day after the operation.

The suprapubic wound should cease to leak urine during the fourth week, and should be healed during the fifth week.

I have performed prostatectomy by suprapubic enucleation nine times, and all the patients recovered from the operation. Eight of them left the hospital or nursing home cured of their obstruction. One died suddenly on the tenth day after operation from a profuse hæmorrhage into his stomach from an aneurysm of the splenic artery, which was laid bare in the floor of a chronic gastric ulcer. His urinary condition was perfectly satisfactory at the time, and he was up and out of bed for the first time when the accident occurred.

Eight of my cases were for prostatic hypertrophy and one for a small fibrous prostate. The latter was the most difficult operation of its kind I have performed, but the result was perfectly satisfactory.

In three of the nine cases calculi were also present.

The operation of suprapubic prostatectomy is one of the most satisfactory in surgery, and there is little doubt that the near future will see its general application to all well-marked cases of prostatic obstruction.

August 13th, 1906.

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CORNS.

By J. FOSTER PALMER, M.R.C.S., L.R.C.P.

Read before the Chelsea Clinical Society.

GENTLEMEN,—For bringing forward so apparently trivial and unimportant a branch of clinical study I need not apologise, for John Hunter did not consider it beneath his dignity to write about it. But some excuse is needed if I have to dissent in any degree, as I may, from the views held by so great a student of anatomy.

"So long as all the conditions remain the same," says Paget, speaking of hypertrophy, "each part of the body retains its state, but when the conditions alter so that a part is more than usually exercised in its office, then it manifests a power of renewing or accelerating its growth. It is as if each healthy part had a reserve power of growth and development, which it puts forth in the time of emergency."* In case of increased action of the skin, for example, the waste of tissue is increased, but the epidermis "does not grow thin; nay, it grows thicker, to protect the cutis; it puts forth, as it were, a reserve power, which is enough not only to repair all amount of waste within certain limits but to increase the quantity of tissue to the amount required."†

If this be so, it is clear that clavus is not the result of pressure alone. There must be something else. In other words, tight boots won't cause corns. You might, for instance, lie in bed for twelve months with tight boots on and get no corns, whatever else you might get. Paget, it is true, says that pressure causes hypertrophy,‡ but this seems inconsistent with his statement quoted above, if the words are taken in their obvious sense. He further says that constant pressure causes atrophy, but that occasional or intermittent pressure causes hypertrophy.§ I submit that even intermittent pressure is not necessarily friction, and that it is the friction, not the pressure, whether constant or intermittent, which produces corns.

John Hunter takes a somewhat different view, which, however, does not seem to include all the facts any more than that of Paget. He says that pressure from within causes atrophy and pressure from without causes hypertrophy.* It is difficult to see how the one can exist without the other—how, in fact, there can be pressure without resistance. Hunter says, "A corn is a thickened cuticle arising from external pressure, which is præternatural and excessive."† But experience seems to show that pressure alone tends to produce absorption and atrophy, friction to produce hypertrophy. Are not bed-sores examples of the former condition? It is pressure, not friction, that causes the skin to disappear. This is especially evident in cases of paralysis, where increased action is impossible. The latter condition, hypertrophy, within certain limits is protective, but when excessive it becomes a morbid growth. When it is physiological it is called a callosity, when pathological a corn. A callosity is not a corn, though they are often spoken of as if they were synonymous. Tylosis is one thing and clavus is another. Tylosis is protective, clavus is destructive. In tylosis there is no atrophy, no destruction of the cutis. In clavus the indurated cuticle, growing in both directions, presses upon the underlying corium, and in many cases pierces completely through it and presses on the tissue beneath.‡ The growths on the finger-tips of the left hand of the violinist are callosities, and enable him to play better. There is plenty of pressure here, and intermittent pressure at that, but the friction is only occasional and not continuous, so that there is no morbid growth. If the friction and pressure were continuous it is hardly conceivable that he would be able to practise ten, or even six, hours a day. There would appear to be sufficient friction and

* 'Lectures on Surgical Pathology,' Turner's edition, 1863, p. 48.

† *Ibid.*, p. 49.

‡ *Ibid.*, p. 65.

§ *Ibid.*, p. 66.

* 'Surgical Works of John Hunter,' Palmer's edition, 1835, vol. iii. p. 466.

† *Ibid.*, vol. i, p. 561.

‡ Tylosis is, of course, derived from the Greek τύλος, a callosity produced by labour, or an enlargement or protuberance generally. Clavus, on the other hand, is the Latin *clavus*, a nail. A corn, at its worst, may be fairly taken to represent a foreign body, such as a nail, driven in through the two layers of skin, and penetrating to the tissue lying beneath it.

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intermittent pressure to produce the superficial hypertrophy, but not enough to cause atrophy

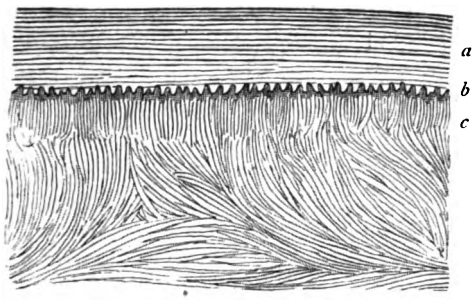

FIG. 1 A.—Normal skin; *a*, cuticle; *b*, rete mucosum; *c*, corium.

beneath. With the harp it is different. There is both pressure and friction, and the liability to corns must be far greater.

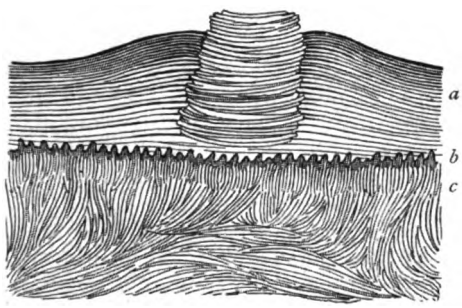

FIG. 1 B.—Tylosis; *a*, cuticle; *b*, rete mucosum; *c*, corium.

Rindfleisch says that corns and callosities are both caused by external pressure, but that "The difference in the results depends on a variation in

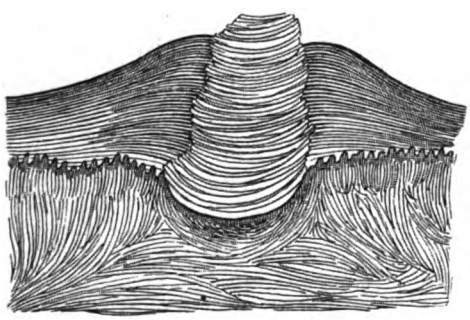

FIG. 1 C.—Clavus; *a*, cuticle; *b*, rete mucosum; *c*, corium.

its mode of action; if the point at which the two opposing forces come into collision coincides accurately with the point of contact between the compressed part and the compressing part, a callosity is produced; if, on the other hand, the two points do not coincide, if the seat of conflict is withdrawn into the interior of the compressed part—in other words, if the compressed part is capable of yielding to the pressure—a corn results."*

So we see that even in so simple a matter as corns there are at least three different views among pathologists as to their origin and causation.

But if this view is correct, if external pressure is the efficient cause in both these conditions, why is the epidermis hypertrophied and the corium atrophied? Both are subject to pressure. On the other hand, it is friction to which the epidermis is subject, and from which the corium is protected. The constant attrition against the convolutions of the boot-leather, rendered still more severe and irritating by the counter-pressure from the extremities of the bones, often displaced by ill-fitting boots, causes increased action in the cuticle. The "more than usual exercise in its office" which Paget postulates† takes place, and the expected result follows: "It grows thicker—it puts forth a reserve power, which is enough, not only to repair all amount of waste within certain limits, but to increase the quantity of tissue to the amount required."‡ Only in this case, unfortunately, it goes beyond that. It does not know when to stop. *Hinc illæ lachrymæ.* It outsteps the bounds of physiology, and enters the region of pathology. Its successful growth makes it aggressive. It gets big and becomes a bully. Now, instead of being the friend and protecting shield of the underlying derma, it becomes its deadly enemy, and annihilates it. The derma, on the other hand, is not subject to friction, only to pressure. It feels not the wobbling of the loose, uneven boots. It only feels the pressure, usually intermittent, transmitted through the overlying cuticle. Therefore it atrophies. There is no friction, otherwise "increased action," to stimulate it to the acceleration of growth necessary "to increase the quantity of tissue to the amount required." The sensitive and now unprotected papillæ are pressed upon by the enlarged and hardened epidermis, causing acute pain, and the corium lying directly underneath the tumour

* 'Manual of Pathological Histology,' Baxter's translation, New Sydenham Society, 1872, vol. i, p. 357.

† Paget's 'Lectures on Surgical Pathology,' p. 48.

‡ *Ibid.*, p. 49.

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may completely disappear. The hypertrophied cuticle will then be in immediate contact with the subcutaneous cellular tissue, or lie on a joint or sheath of a tendon, in which case a bursa usually results (Fig. 2).

Why a bursa should form I cannot pretend to explain. It is very easy to say that it is a provision of Nature to protect the underlying parts, bone or tendon. But this is the result, not the cause, and in no way helps to explain the *modus operandi*. We assume a sort of beneficent object in an unknown imaginary force, and are content to leave it at that; but this is merely a cover for our ignorance as to the actual factors by which the result is brought about. The bursa appears to be just a

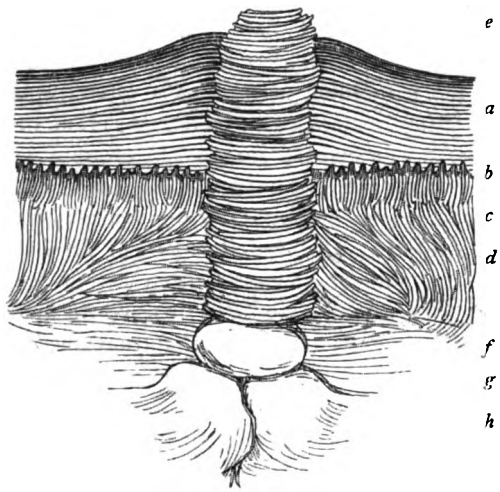

FIG. 2.—*a*, Cuticle; *b*, rete mucosum; *c*, corium; *d*, subcutaneous cellular tissue; *e*, hypertrophied cuticle = corn; *f*, bursa; *g*, joint distorted by ill-fitting boot; *h*, heads of bones separated from hypertrophied cuticle by bursa.

further development of the enlarged cuticle round some of the spaces in the subcutaneous areolar tissue. But why should it take this particular form? And why should Nature, having allowed the corn to torture and actually destroy the corium, suddenly become so very careful lest it should injure the joint or tendon beneath it? I am inclined to doubt its beneficent intentions, and to suspect that it has some sinister designs on these structures after all. I submit, therefore, that it is well not to put too much faith in bursæ, but to endeavour to restore both corn and bursa to their normal condition at the earliest possible opportunity.*

This leads us to the question of treatment. The treatment of uncomplicated corns—and it is these I am describing—is comparatively simple and practically certain in its results. But there is a treatment to be avoided as well as a treatment to be followed, and it is to the former that the complications are generally due. This is, indeed, the principal difficulty, to dissuade the patient from cutting his corns, or from going to a chiropodist to have them cut. The greatest sufferers from corns are those who have them cut periodically. They get, perhaps, a little temporary relief, and then continue to live in greater torment than before till the process is repeated. To such persons life is a misery. Can anything be more unscientific, more opposed to common sense even, than to attempt to cure a corn penetrating the cutis by slicing off the outside of it—that is, the least abnormal part? For in its external layers a corn is little more than a callosity, which is almost physiological. When thus treated the pressure and friction are only transferred from the centre to the circumference. The hardened cuticle, deprived of its outer layers, is more sensitive than before, the papillæ being more exposed; the area of induration is increased, and the last state of the patient is worse than the first—that is, if the causes of friction are allowed to continue. If they are removed, the corn will disappear of itself and no cutting is necessary.

When the corn is more deeply cut certain further structural changes ensue. Instead of the horizontal flattened layers of the thickened epidermis, we now have layers perpendicular to the surface of the skin, and covering a number of elongated and extremely sensitive papillæ with enlarged extremities. These enlarged papillæ are the result of hypertrophy of the upper surface of the corium which has been exposed to friction by removal of the hardened cuticle above. This condition is, in structure, identical with a wart, and only occurs, I believe, when cutting has been resorted to. (Fig. 3.)

* There is one factor in the production of corns which is usually lost sight of. I refer to climate. I am told that in the warm, moist atmosphere of Ceylon corns are practically non-existent. Whether this is really due to the climate or to the fact that people do not require to wear boots I am unable to say, but I understand that all indurations of the skin, including, I believe, keloid, are at least modified by it. I have no information on the point with regard to other tropical countries.

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Another common result of treatment is hæmorrhage, which may give rise to various further troubles. If the corn penetrates the cutis, the knife must enter the subcutaneous tissue in order to reach its apex, and in this case hæmorrhage is almost sure to follow. I doubt if hæmorrhage ever takes place in corns without cutting. It is usually slight, of course, but in view of possible sequences is obviously better avoided. In any case, if a knife has to be used, it should be with the aseptic precautions of the surgeon, and not by the empirical methods of the chiropodist. Inflammation, when it occurs, usually comes from the use of strong caustics, but it may also follow cutting. Suppuration, too, may result from either method. They rarely occur except as a result of injudicious treatment.

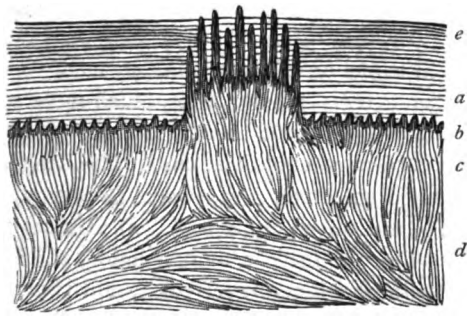

FIG. 3.—*a*, Cuticle; *b*, rete mucosum; *c*, corium; *d*, areolar subcutaneous tissue; *e*, abnormal development of epidermis on enlarged papillæ after cutting = wart.

Corns are, in the true sense of the word, a luxury.* They are certainly not a necessity of life, and no one need indulge in them unless he chooses. If boots fitted accurately, and only moved in harmony with the feet there would be no corns. It is the opposing forces which cause the mischief, the boots moving one way and the feet the other, and even then they only occur when there are internal projections in the boots. These inequalities may be greatly reduced, if not altogether removed, by using lasts when the boots are off, and padding the inequalities with wool when they are on. Indeed, I believe that if all people wore good thick woollen socks instead of cotton or silk abominations, there would be few, if any, corns extant.

This, however, is not strictly the matter before us, which is the treatment, not the prevention, of corns. Our patients do not ask us how to prevent corns. They never consult us until they have them, and not often then. They will, in fact, do anything first. They will cut them, sometimes with fatal results. They will go to a chiropodist. In fact, they will undergo any amount of torture, any amount of risk, either at their own hands or at the hands of others, and will continue to suffer for any length of time, rather than follow what would seem to be the most simple and obvious course—viz. to consult a surgeon and undergo a short and painless cure. Probably the worst corn that ever existed could be cured in a fortnight, provided, of course, that it had not become complicated by improper treatment. But patients (and patients they are truly!) prefer to suffer a considerable amount of pain and expense to make them permanent.

I believe that all corns, if they are nothing more than corns, are perfectly amenable to treatment. The only difficulty is that patients will not allow us to treat them. Removal of the causes of friction will, in a large number of cases, result in complete recovery without any further application, but usually saturation for a longer or shorter time with water or glycerine may be necessary in addition. The length of time it is required will depend on the severity of the disease. But this treatment may, be it remembered, in some few cases, especially of corns on the sole of the foot, involve staying in bed, or at least lying on the sofa. This is, perhaps, the chief difficulty. People will not go to bed to cure corns. If they did, they could confidently expect to get rid of them, however bad. But they prefer to wait till they are ill. An attack of typhoid, or even smallpox or scarlet fever, will cure any corn in the world. But until they get the attack they decline the treatment. And they may never get the attack. Such cases, however, are extreme and rare, so that it is seldom necessary to resort to such measures. There are, in fact, very few corns that will not succumb to a nightly bread-poultice or water dressing, with a soft perforated plaster of spongio-piline by day, to avoid friction. If the perforation can be filled with glycerine it will hasten the cure by keeping the hardened skin saturated by day as well as by night. Still, the all-night poultice or water dressing will usually suffice, and very often much less than this. Soaking the feet night and morning in warm water,

* From the Latin *luxus*, dislocation or excess.

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with or without the addition of sodium carbonate, will frequently put an end to corns. Indeed, Dr. Evershed says that he has constantly cured them by fifteen minutes' soaking twice a day. This was in sea-water, but the principle is the same, although the addition of alkaline salts, as in sea water, or of boric acid, would doubtless hasten the result. The action of the alkaline carbonates on the skin is well-known, and is exemplified in the case of bottle-washers, who use sodium carbonate, and whose nails are softened and made thin and flexible by it or even sometimes almost destroyed. The hardened cuticle of the corn may be thinned and softened in the same way. Still, ἄριστον μὲν ὕδωρ. Water is, after all, the best and safest cure, with patience added. The difference in the treatment of different cases consists, not in the nature of the remedy, but in the length of time during which it needs to be applied. The old method, suggested, I believe, by Fergusson some sixty or seventy years ago, is still available, and we have not got far beyond it in our present practice. "Poultice till they drop off" may not be literally applicable to every individual case; but by the principle, interpreted intelligently, you may cure them all. It is, in most cases, only a question of time. The particular method is unimportant; the general principle underlying it is essential.

I need not say that in thus dogmatising about corns I refer to simple, uncomplicated corns only. The complications, as I have already said, I believe to be in nearly all cases the result of injudicious and inappropriate treatment. When from cutting or any other cause suppuration takes place, we are dealing, not with a corn, but with an abscess, and this abscess will require incision and antiseptic treatment. This treatment, however, will probably cure the corn as well. Inflammation, also, if not so extensive as to be dangerous, will necessitate the salutary rest and treatment the patient would not otherwise have submitted to, and thus, though in itself an evil to be avoided, may ultimately have beneficial results. The warty growths, too, above described, are not of the nature of simple corns, but are usually the result of deep cutting. In some cases they are amenable to the treatment by continued soaking, but in others they may require excision or the application of glacial acetic acid as in similar growths elsewhere.

August 13th, 1906.

THE EAR, NOSE, AND PHARYNX. By ST. JOHN ROOSA and BEAMAN DOUGLASS. (MACMILLAN & CO., LTD.)

THE laudable object of this well-written book is to enlighten the practitioner on those earlier conditions of departure from the normal in the throat, nose, and ear that are so readily amenable to treatment, and which are so often left to drift and develop into serious affections. The directions for the examination of those special regions are short and practical, and too much space is not taken up with unnecessary minutiæ with reference to ear tests, although the essentials are clearly given. Diseases of the ear are very well and fully described, and special attention is given to the important and in every-day medical work often embarrassing subject of foreign bodies in the ear. This is a subject that is often very scantily discussed in works on this region.

While brevity is commendable, much more attention would have been desirable on the all-important subject of cholesteatoma. The dangers of this change are great, and always imminent, and cannot be too much emphasised. The treatment of cholesteatoma of the ear is often left in the hands of the medical attendant for years, and it is therefore essential that the guiding principles should be made plain. To syringe with a watery solution of bicarbonate of soda, as recommended by the authors, is most reprehensible. The only safe treatment is to instil spirit drops over a long period, and watch for any urgent symptoms and operate immediately on their appearance. The surgical operations are carefully and tersely reviewed, and the various indications for operative interference pointed out. The authors favour considerable temporising in acute conditions of otitis media and mastoiditis. They rely overmuch on leeching and other applications. In skilled and practical hands early operation is safer, and is in str.cter accord with scientific principles. Many good practical hints and numerous useful recipes are given in this work, which cannot fail to be helpful to the practitioner. We can safely recommend it as a guide for elementary study.

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THE CLINICAL JOURNAL,

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

SOME CASES OF CANCER.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—It occurred to me when I was suddenly requested by a colleague to take his place to-day that some cases of carcinoma which are in my wards would serve as a text for a clinical lecture, and especially because each one of them raised a point, or points, of very great importance with regard to the disease.

The first patient that I propose to refer to is a compositor. His age has little bearing upon the nature of his disease except this, that carcinoma, which he has, is one of the privileges of seniority. You must not, however, suppose for one minute that the youthful may not have carcinoma. That curious disease carcinoma of the vermiform appendix has not infrequently been met with in quite young people, and has almost always been—until it was microscopically examined—supposed to be tuberculosis. Now, this patient came with an ulcer at the posterior part of his rectum and anal canal. Some portion of it was removed and microscopically examined, and was found to be columnar celled carcinoma, undergoing, by-the-by, mucoid change, which is common in carcinoma of the columnar-celled type, which grows in mucous canals. I attached no importance to that, because having watched cancers which have been undergoing mucoid change, and also cancers which have been undergoing colloid change, I have not myself observed that it ever indicates any increased degree of malignancy. Now, this carcinoma was also examined under an anæsthetic before any attempt at removal was made. The examination under an anæsthetic was made, of course, to ascertain its

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extent. I should not say because the finger could not be introduced beyond a carcinoma of the rectum that therefore that carcinoma was outside the possibilities of removal. But obviously if a surgeon cannot get his finger beyond a carcinoma of the rectum he must of necessity remain in ignorance of its upper extent, and when the operation is attempted it may prove to be outside the possibilities of his removal. However, this was an easy case, because the finger easily passed beyond the carcinoma. Further, the finger was unable to detect any enlargement of the glands in the sacral hollow, because as far as we could tell the carcinoma was not of very long duration, and because we knew that that carcinoma was growing in the interior of a muscular canal. I think I have said before that the carcinoma growing inside the human bladder, carcinoma growing inside the human intestine, carcinoma growing inside the human stomach, and carcinoma growing inside the gall-bladder, usually take a long time escaping to the exterior; but these are more favourable than the circumstances when carcinomata are those growing from the mammary gland or tongue and the pharynx. I shall presently refer to carcinoma growing in the mammary gland and carcinoma growing in the tongue, and you will see the rapidity with which they spread into the neighbouring lymphatics. Our examination of this condition under an anæsthetic gave us clear light upon another point, and it was this: that not only could the finger be passed beyond the tumour in its upper limits, but it also could easily be passed on either side, showing that the growth had not yet extended into the ischio-rectal fossa, and therefore it was determined to remove this carcinoma. But now another very important point in the case had to be taken into consideration, because it was found that upon examination of the urine it contained a very considerable quantity of sugar. The largest quantity of sugar that the urine contained at one time was almost 1200 gr. of sugar *per diem*, and that was contained in a very considerable quantity of urine, nearly four pints. If you make a passing calculation of the amount of sugar that has been excreted in the urine, you will find that it amounts to nearly three ounces in the twenty-four hours. And if you care to make a further small calculation, you will find that it amounts to nearly one pound in the four days, which is a very considerable quantity to lose.

Experience has shown that major operations performed upon diabetics are exceedingly dangerous. I remember some time ago being called upon to crush a stone for a patient who, upon proper examination, was found to have a large quantity, 3 per cent., of sugar in his urine. The stone had to be crushed because it was clear that if the stone was not got rid of the patient could not continue to perform the duties of life, and his life would be a burden to him. Crushing was obviously, in his case, the proper course to pursue, because, although he was seventy years of age, his prostate was but moderately enlarged, and in every other respect he was in vigorous health. Moreover, one knows that wounds in diabetics are very apt to become septic, and even gangrenous. It is supposed that the ordinary septic bacteria are able to grow and flourish better in tissues which are impregnated with sugar than in tissues of a healthy person. However, a consultation with a physician was held upon this point, as to whether it was right for this patient to take the increased risk of lithotrity. The conclusion we came to, after considerable inquiry and consideration, was this, that provided the sugar was under control, the operation might be undertaken with a reasonable prospect of success. It was found when the patient was put upon a strict diet that the sugar almost entirely disappeared from his urine. So the stone was crushed and the patient did exceedingly well, and in about a week's time he was up and walking to his room and on ordinary diet, but of course on no starches or sugar, and he was making his arrangements for going home. But the weather turned exceedingly cold suddenly, and one day when I went to the Nursing Home I was informed that he was rather sleepy and very quiet. Next day he became still more sleepy and more quiet, and afterwards became comatose and died of his diabetes. I do not know how that calamity could have been prevented. We did all that we could. But he probably got the condition known as "acetonæmia." I believe it is customary to give people with diabetes who are threatened with acetonæmia considerable quantities of carbonate of soda in their milk or whatever fluid they are taking. However that may be, I knew from my own experience that an operation of moderate

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severity upon a diabetic was a very perilous proceeding. We therefore set to work with the person whose case we are particularly discussing and got the sugar in his urine under control, and I think I am right in saying from memory that the amount of sugar which he was excreting in his urine had been reduced before the operation from 1188 or 1200 gr. *per diem* to 36. The operation was the ordinary straightforward cutting operation, and care was taken to guard against serious infection. I must say that operations upon the rectum, to my mind, have been rendered very much more safe, and very much less painful, by the adaptation of proper rectal packing and drainage after the operation. For instance, in this particular case every possible precaution was taken to perform the operation with as much approach to asepsis as possible, and after it was over the cut surface was dusted with iodoform, a drainage-tube passed into the rectum, and the wound was steadily packed with a quantity of iodoform gauze. In consequence of this, any septic mucus or septic fluid in the rectum could pass out of the drainage-tube and was not brought into contact with the newly-severed tissues. By the time the drainage-tube was due to come out, on the third or fourth day, all the lymph-paths and cut surface of the wound had become sealed more or less securely by the usual process of inflammation, and the patient, I am correct in saying, never had any serious degree of sepsis, or I believe even discomfort, after his operation. I observed the day after operation that the temperature was 99° F., and then it fell to 97° F. and remained there, and later on it rose a little. So our efforts in that direction were successful. Now I come to another point with regard to this man. When we made our examination of his rectal growth by the usual microscopical process we found that it was a columnar-celled carcinoma. I will just show you by a diagram my own crude conception of an ordinary rectal or tongue carcinoma, or of a carcinoma of some mucous or cutaneous surface of the body. First of all, I will put on the board a simple papilloma of the rectum with Leiberkühn's glands—that is to say, merely an elongation of the interglandular processes, with a covering of epithelium and central connective tissue and blood-vessels and lymphatics and nerves. I will now complete that simple papilloma into carcinoma of the rectum by making the epithelium spread down into the tissues. Mr. Dive has kindly put up specimens which show clearly the intertubular papillæ growing into the rectum, and you can also see the intertubular or interpapillary epithelium growing into the wall of the rectum, first into the sub-mucoid and then into the muscular coat, and then outside the muscular coat into the surrounding tissues, the bladder, the ischio-rectal fossa, or the sacrum, or whatever the nearest structure may be. There is nothing at all singular about that. Observe, if that is the whole truth, that that man's future prospects, from my point of view, are not bad. He has got his carcinoma inside the rectum, he has the muscular walls of the rectum between him and the tissues on the exterior. That type of carcinoma does not seem very prone to spread along the lymphatics into the neighbouring lymphatic glands. That, I think, so far is favourable for the man. But now, when we come to look at this large section, we discover that there is something else proceeding, because at the margin of the rectum you see the squamous epithelium of the anal canal. Now we observe something very strange is going on in this, because here, again, the interpapillary processes are growing inwards into the deeper tissues. Surely he has squamous epithelium growing into his tissues, as well as columnar epithelium. If that is true it is a very striking fact, because he has two types of carcinoma proceeding in his body at the same time. What does that mean? Should we look upon epithelioma of the tongue as one type of carcinoma, and the carcinoma of the mucous surface covered with columnar epithelium as another kind of thing altogether? Presumably in this case both types of carcinoma are due to one cause? What is that? There is a little point in his clinical history which seems to me, at all events, to throw some light upon it. So long ago as February, 1898, he had some anal discharge, and he has had anal discharge ever since, so that he has had some trouble there for many years. I have seen many people who have had a long unhealed wound ultimately become cancerous. There was a man in Harley Ward the other day who had a granulating wound of the thigh for eighteen years or more, and at last it became cancerous. I remember a man who had an old ulcer on the dorsum of his foot for twenty years, and that became cancerous. And how many

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people have you not seen who have had syphilitic ulceration of the tongue which at last became cancerous. If a human being has an open sore long enough it at last may become cancerous. This man has had some cause of trouble there for a very long time: there has been some open sore. There are other points in his history, which, however, I do not think have any great bearing on his present condition. There is reason for a strong supposition that he has had syphilis, but I think that would only act as a chronic syphilitic ulcer of the tongue would act, and that is, as a portal through which some infection would enter.

I wish now to draw your attention to another point. I myself examined that man and the growth in his rectum, with the rooted conviction in my mind that it was columnar-celled carcinoma, and of course took great pains to examine the channels which drew their lymph from that columnar-celled layer—that is, the glands in the hollow of the sacrum. But the cutaneous surface of the anal canal does not send its lymph into the hollow of the sacrum, but along the lymphatics into the perineum along the fork by the side of the scrotum, in the lymphatics of Scarpa's triangle and along Poupart's ligament and through the femoral canal into the lower pelvic glands. He has been felt with the view of ascertaining what the glands in the groin are like, and one significant thing has been noted, namely that those glands are slightly enlarged. They require careful watching to see if they remain enlarged. Should there be the slightest sign of increase, in spite of his diabetes it will be necessary and right to do another and rather considerable operation for the removal of the glands. It will be reasonable soon, and I suggest it would be better as soon as he can bear it, to ascertain whether they contain inflammatory tissue or carcinoma. I never believe such a gland is inflammatory until I have seen that it is.

Leaving that case now, which you will admit raises many important points as well as interesting ones, I will proceed to consider another, namely one in Kenton Ward. This is a man who had quite an ordinary history. He had for a time felt that he must spit a little more than he had been accustomed to. Then at length he felt a sore place, a painful ulcer, at the back of his tongue, and later an ulcer appeared. At last he came to this hospital with a tumour on the back of his tongue, with a small, irregular ulcer in its centre. Now, for clinical purposes we may divide the tongue into thirds—anterior, middle, and posterior third—very much as, in the anatomical department, we are accustomed to divide the parts we describe. And this cancer was situated in the posterior third. The history said that the man had had the carcinoma for three months; but the carcinoma itself was not very large. Observe that I have already said to-day that if a person has carcinoma in any organ which does not possess a well-marked capsule, that carcinoma will spread with extraordinary rapidity into the neighbouring lymphatic glands, and therefore it became necessary in this case to at once begin to investigate the neighbouring lymphatic glands. For what is the use of removing carcinoma of the tongue and leaving the carcinoma growing in the neighbouring lymphatic glands? To ascertain the condition of the lymphatic glands it is necessary to know where you are to find them, and the lymphatic glands for the tongue, as I described in a lecture which I delivered in this theatre, and which is published in the CLINICAL JOURNAL for June 8th, 1904, are rather curious. You cannot reason about lymphatics at all, you can only observe the course of lymphatics. Nor is it very easy to ascertain the course of lymphatics by the anatomist's method of squirting coloured fluid into the lymphatics and lymph-channels. It is not a very searching method of examination. The results of disease are to me very much clearer evidences, and they supplement the results from the best method of injection. I shall draw a rough diagram of the tongue on the board, and you will see that we can divide the region into thirds. The lymphatics at the tip of the tongue open into the submental glands and those in the submaxillary triangle. If a person has epithelioma at the tip of the tongue, which we not infrequently see, do not forget to feel underneath the mental process of his jaw for lymphatics there. They are not very easy to feel, and do not trust your sense of touch, but when the time comes to perform the operation do not hesitate to look and see whether they are or are not involved. I shall come to that point again. The lymphatics from the middle of the tongue empty into the glands in the floor of the mouth first. These glands are certain to be overlooked if you do not know how to feel for them. Put one finger inside

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the mouth and the other outside, and you will feel them slipping between your fingers. In addition, the glands at the middle of the tongue open into the glands which are underneath and within the substance of the submaxillary gland. The anatomist talks about the glands on the surface of the sub-maxillary gland, but he often forgets the glands which are situated inside that gland. So with carcinoma of the middle of the tongue: it may be necessary and wise to remove the whole submaxillary gland for fear of leaving a cancerous gland in the centre. I shall now tell you something about glands which constitute a trap into which we may fall. Here is a lymphatic vessel, making a tour down the neck, and ending in a gland which is opposite the cricoid cartilage. An enlargement of this gland so far down the neck might easily be overlooked. The lymphatic tract is mentioned in some books on anatomy as having been ascertained by injection, but it has been longer known to surgeons as a fact by observation. So that in an operation to take away these glands it is necessary to make an incision which will go to the symphisis of the jaw to deal with these symphisial glands, and the incision has also to be carried down to the level of the cricoid to get at the gland which is opposite it. It is an incision which also serves to secure the rest of the glands when a flap is turned back. We have a different condition of things in the man that we are speaking about. The cancer is situated in the posterior third of his tongue, and the infection would pass directly outwards and open into the lymphatics underneath the angle of the jaw, sometimes called the tonsillar glands. Some of them are hidden away under the lower part of the parotid gland. When we came to examine this patient you can imagine with what interest we all sought for enlargement of the gland underneath the angle of the jaw over the carotid vessels. The man owns to drinking two quarts of beer a day, so he is well supplied with adipose tissue, and everyone felt the force of the note which Mr. Viner made—I forget the exact expression but I think it is "a suspicious fulness." But still, that means nothing, it is a way of hedging. I myself thought—I only thought—I could feel something of the gland underneath the angle of the jaw; perhaps I was a little more dexterous at feeling than some, perhaps I rolled the gland a little more skilfully under the tip of the assiduously trained index finger. Still, my opinion that I felt it does not go for much. Whether you felt it or not, I am absolutely certain of one thing, and that is that I should not hesitate to look at that group of glands. And when we did so in this case we found an enlarged gland underneath the angle of the jaw and several over the course of the carotid vessels, and those glands were there and then microscopically examined and found to contain carcinoma; in fact, they contained masses of squamous epithelium exactly the same as those which you might feel growing into the tongue. All this is very striking, and I am very anxious about the future of this man. I will tell you why. The carcinoma is close to the tonsil, which is surrounded by lymphatics, which again communicate with the lymphatics at the back of the pharynx. These, of course, cannot be removed, but I did the best I could. Another thing is, that the lymphatics of the tongue have this peculiarity. There is a network across the dorsum of the tongue which carries the lymph from one side of the tongue to the other. And in this theatre I have mentioned the case of an old lady who had a small carcinoma removed from the back of the tongue. She had no glands on that side—that is to say, on the same side as the carcinoma—that could be felt; no incision was made. She came again a year afterwards, and she then had carcinoma of the tonsillar glands underneath the right angle of the jaw. Originally her trouble had been on the left side. It had passed across the dorsum of the tongue, and it may have passed behind her pharynx into the lymphatics of the opposite side of the neck. So you will see that this man has not got a very pleasant future before him. With this in my mind, I removed half his tongue, and removed his carcinoma very widely behind, and tried to remove the tongue down to the epiglottis and to remove the anterior pillar of his fauces a long way down. In doing that I noticed several things which rather startled me, but it is worth his while to take considerable risks in the hope of attaining future safety. So much for the man, whom you may see and study, or, if he is not there, he is sure to come to Kenton again for observation.

Now I will come to another case, which was operated upon a short time ago. She is in Lucas Ward. And she again raises another very important question concerning the spread of carcinoma,

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and incidentally concerning the proper methods of operating for it. In August last year a very stout woman was sent up from Hampshire with a carcinoma of the left breast. Speaking from memory, I believe I am correct in saying that she had had the carcinoma for at least two years, and I remember that the medical man, my friend who sent her up, wrote to me at the time to explain that it was not his fault that the case had been allowed to proceed so far. I did at the time the usual operation, and removed, I trust, the whole mammary gland, which is a huge structure. I removed both pectoral muscles, and I did my best to remove all the axillary fascia and lymphatics. I went above and I went below the upper part of the axillary vein, and I think I left no trace of carcinoma below the clavicle. We had no reason whatever to suppose, from our clinical examination, that there was any trace of carcinoma above the clavicle ; and yet why you or anybody else should for one moment suppose that the clavicle is the slightest bar to the spread of carcinoma into the neck I cannot imagine.

Now comes the latest act in the drama, because lately the patient returned, and it was then quite easy to feel enlargement of the lymphatic glands in the posterior triangle of the neck. The carcinoma had obviously spread into the lymphatic glands in the posterior triangle of the neck. One of its methods of spread might have been this. The lymph-channels pass underneath the greater and lesser pectoral muscles into the axillary glands and upwards, over and under the vein, into the glands at the posterior triangle of the neck. I suppose that is the usual way, and that is the way we hope it goes. But that is not the only way. Lymphatic channels do not always go by the longest and most inconvenient route, they sometimes go by the shortest and most convenient route, and in the case of the mammary glands there are lymphatics which pass from the mammary gland over the clavicle and into the subclavian glands without going through the axillary at all. I remember once a patient came back after she had had her breast removed, and she had an enlarged lymphatic gland, but it was in a very unusual place, namely in the upper aperture of the thorax, underneath the attachment of the sterno-cleido-mastoideus. I guessed that the cancerous lymph in that case did not pass through the axilla or underneath the clavicle, because then it ought to have infected the subclavian glands themselves. I think that in that particular instance the lymph from her breast went up by a channel which short-circuited itself into the gland underneath the sterno-mastoid. When I came to operate upon the patient now in Lucas, by a triangular flap being turned inwards from the posterior triangle of the neck towards the middle line, cancerous lymphatic glands were found in the posterior triangle of the neck, and these were removed without any particular difficulty. And it was then found that she had other lymphatic glands which could be neither seen nor felt before the operation, and which were adherent underneath the sterno-cleido-mastoideus. After some trouble, and with some trepidation, these glands were removed. I did not see the thoracic duct ; I suppose I ought to have done so. I saw the layer of fascia covering the phrenic nerve, so it was not a very pleasant place to remove adherent glands from. Now, this is the question which is in my mind : Ought I not to have been rather wiser when I operated upon that patient in the first instance and have said to myself this : " Here is a very stout woman who has carcinoma which is known to have existed for two years in the left mammary gland, who is also known to have considerable enlargement of the lymphatics in her arm-pit ; would it not be only wise to go a step farther and carry the incision up into the posterior triangle of the neck to see what the condition of the subclavian glands is ? " Because you cannot see them, for they are covered by the superficial fascia and the platisma myoides and the deep fascia. You cannot feel them with your fallacious fingers, especially as she is very fat. I am sure I ought to have explored the neck, and I think that the omission of that precaution must have shortened her days. I am gradually getting to this stage in my thinking about carcinoma of the breast and of all other regions, and that is that it is becoming more and more necessary not to believe you can feel the lymphatic glands, but to do an exploratory operation to try to see what is the matter with them. What a great comfort it must be to the patient after the operation is over to know that the lymphatic glands have been properly looked at and that nothing has been found in them ! Patients are very thoughtful in these matters, and they will ask, if the operation has been in Scarpa's triangle,

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whether there was any disease higher up; that means inside the abdomen. Mr. Dive has kindly placed on the table a series of sections of the growths of which we have been speaking. Most of them are perfectly straightforward, and I advise you strongly to look at all those microscopical specimens, because the time is coming when the surgeon will have to look at the specimens at the time of operation and form an opinion upon them, and those who have not done this assiduously will be taken at a disadvantage.

August 20th, 1906.

Mutual Relations Between Heart and Gastro-intestinal Affections.—Schmidt relates several case histories of persons who applied to him for relief from gastro-intestinal trouble distinguished by accumulations of gas in the stomach and intestines. These patients were otherwise apparently in good condition and after the gas had passed off in eructations or flatus they were relieved until it assembled again after the next meal. He is convinced that the trouble is due to the insufficient absorption of the gases by the venous blood. Digitalis helps here. Normally the blood absorbs a large proportion of the gas ingested or formed in the digestive tract. When there is any stasis in the gastro-intestinal blood-vessels, the gases are not absorbed, and their accumulation is a sign of defective functioning of the circulatory apparatus, sometimes before any other signs call attention to it. He also describes cases of the reverse condition, namely, functional disturbance of the heart from gastro-intestinal affections. The digestive disturbances are generally mild and of a functional nature, but there are almost always accumulations of gas. The prognosis is favourable so long as the heart trouble is only functional, but if it is organic irreparable harm may be done by reflex action, as in a case described in which a patient with mitral insufficiency succumbed to an attack of acute gastritis. Faradization and massage of the abdomen may prove useful in supplementing the regulation of the diet and bowels. Rest in bed is imperative for all these patients. It has proved particularly useful in cases of heart affections masked by stomach symptoms. Digitalis, possibly given by the rectum, is more effectual than all other measures combined.—*Journ. A. M. A.*, vol xlvii, No. 3.

NEURALGIA AND HEADACHE.

By LEONARD WILLIAMS, M.D., M.R.C.P.,
Physician to the French Hospital; Assistant Physician to the Metropolitan Hospital.

PAIN which follows the distribution of certain nerves is a very common affection. In the form of sciatica, cervico-brachial neuralgia, and some others it is most often due to a fibrositis or inflammation of the white fibrous tissue in the nerve-sheath and its immediate neighbourhood, and is then much influenced by changes in the weather. "Sciatica" is a condition with which patients are very liable to come with a diagnosis ready made—a diagnosis against accepting which we should always be especially on our guard. According to F. J. Smith,* if the pain is markedly worse at night, or if both sciatic nerves are involved, it is tolerably certain that some cause more serious than a mere inflammation of the nerve-sheath is in operation. For it is to be remembered that pain along one or both sciatic nerves may be due to various causes. It may result from a loaded rectum, from uterine and ovarian displacements, from tumours and disease of the spinal cord itself, and such possible factors should always be very carefully and exhaustively investigated before the pain is pronounced to be due to a neuralgia, and treated as such. For this purpose it is important to determine whether the pain is due to pressure, or whether it is caused by some factor in the nerve itself or in its sheath. In the former case the pain will not be sensibly aggravated when the nerve is put on the stretch—it may, indeed, be to some extent relieved by the process; whereas when the mischief is in the nerve or its sheath, stretching will obviously increase the pain. In order to set this point at rest the patient is placed upon his back, and the pelvis firmly fixed against the bed by an attendant. The limb on the affected side, which must be kept fully extended at the knee, is then gently and gradually raised by the examiner until it is at right angles to the couch; this will put the nerve on the stretch, and if no aggravation of pain results, then the cause is to be sought outside the sheath of the nerve; it is not due to a fibrositis, and the condition is not, properly speaking, a sciatica.

* CLINICAL JOURNAL, December 27th, 1905.

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So far as the treatment of sciatica is concerned, it may be said to consist in the cleansing of the primæ viæ, the regulation of the diet, and the application of massage. Dr. Lee* says: "It is in sciatica, of all the neuralgias, that massage has won its greatest reputation. Truly astonishing results have been obtained even when the affection has been of many years' standing, and after every other conceivable means of relief has proved unsuccessful." This coincides completely with my own experience, and since I have used massage perseveringly, assisted in suitable cases by douching and the application of stimulating liniments, I have had no occasion to resort to acupuncture, surgical stretching of the nerve, the actual cautery, blistering along the course of the nerve, and other heroic remedies which are often recommended, and which the tedious nature of these cases so often suggests to the despairing physician. If massage were employed early in all cases, few would become chronic, and if it is persevered with, in cases which have become chronic, even to the causing of marked wasting of the muscles, it will in time always bring about a cure. Some of the anti-neuralgic drugs, which will be noticed presently, may also be used concurrently. Some of them, especially phenozone and butyl-chloral, have often seemed to me to do good in the way of rendering the massage more tolerable. Without massage these drugs may be palliative, but they are never curative, as they are in neuralgia due to causes other than fibrositis.

Among the causes other than fibrositis which produce pain along the distribution of certain nerves the most important are unsatisfactory blood states. That neuralgia is the cry of a nerve for healthy blood is an oft-quoted saying, and certain it is that undue toxicity of the blood, from whatever cause arising, is an important, as it is certainly the most common, agent in the causation of pain. For toxins in the blood act, not only by direct irritation of a nerve, but they also act, as in gout, by causing vaso-constriction, thus depriving the nerve of its due quantity of nutritive material. Burney Yeo† suggests that a factor of an opposite kind is not infrequently in operation, namely that the blood state may give rise to vaso-dilatation, and that it is a kind of local blushing in the neighbourhood of the nerve which determines the pain. However that may be, all that it is necessary to realise is that impure blood is the most important cause of neuralgia, and that it may produce this effect, not only directly, but also by interference with the normal vasomotor mechanism. Another cause, scarcely less in importance, is the existence of an irritant causing fatigue of the involved nerve.

The presence of a neuralgia, then, should suggest (1) a fibrositis, (2) an unsatisfactory blood state, and (3) the existence of an irritant. With the first I have already dealt; there remain therefore the other two to consider. Of unsatisfactory blood states the commonest is surely anæmia. Whether this be due to convalescence from acute disease, or to mere chlorosis, or to deficient coagulability of the blood* or other cause, it is very frequently attended by neuralgia, more especially about the head and lower part of the trunk on one side. The treatment of such cases resolves itself into the treatment of the anæmia by suitable hygienic, dietetic, and medicinal means. So far as hygiene is concerned, an out-of-door life in a bracing climate is strongly to be advised; the diet should be generous, including meat foods and wines, preferably a good Bordeaux or Burgundy, and plenty of fats. Fats seem to be concerned in some very special manner with the nourishment of the nervous system, and in the form of butter and cream they may be freely given to such patients as we are considering. The best medicine is undoubtedly iron, but the stronger salts, the sulphate and perchloride, are much less efficacious than the citrates and tartarates. The latter are readily assimilated, whereas the former are very apt to upset the stomach. A useful formula is as follows:

℞. Ferri ammon. citrat. . . gr. x.
Liq. arsenicalis. . . . ♏ij.
Inf. quassiæ . . ad. ℥ss.
M., sig.: Ter die post cib.

As strength improves it may be desirable to substitute the following:

℞. Ferri quin. citrat. . . gr. xx.
Liq. arsenicalis . . . ♏v.
Tr. nucis. vom. . . . ♏iv.
Aq. aurant flor. . . ad. ℥ss.
M., sig.: Ter post cib.

* Hare's 'Practical Therapeutics.'

† 'A Manual of Medical Treatment.' See also F. A. Hare, CLINICAL JOURNAL, etc.

* Ross, 'Lancet,' January 20th, 1906.

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The presence of the quinine, even in such doses, may help to subdue the neuralgia. When giving iron in any form and for any purpose it is of the utmost importance to keep the bowels open. This is best done by means of aloes at first (because this drug enhances the effect of the iron) and, later, by cascara. A daily morning dose of a natural mineral water is also very useful.

But where the neuralgia is an obtrusive feature of the condition it is generally necessary to prescribe something which has a direct influence upon the pain when it occurs. In anæmic cases, and in all those in which defective nutrition is pronounced, I have had better results from quinine and gelsemium than from any other combination.

℞. Quin. mur. . . . gr. v.
Acid hydrobrom. dil. . . ♏xx.
Tr. gelsemii . . . ♏x.
Aq. chloroformi . . ad. ʒss.

M., sig.: Every twenty minutes till pain ceases. Not more than four doses to be taken.

This mixture, like most others where the relief of pain is concerned, acts better in small doses repeated at short intervals than in single large doses.

The state of the blood in goutiness is such as to be highly provocative of neuralgic pains. Inasmuch as this condition and its treatment has been previously considered,* it is here only necessary to emphasise the advisability of examining for high arterial tension in all cases of neuralgia, and where this is present to point out the value of iodide of potassium in the treatment of the accompanying neuralgia. As this drug is the most potent of all others in counteracting the gouty state generally, a gouty neuralgia ought very readily to yield to the measures proper to the treatment of the blood state itself. Occasionally, however, it does not, and then it is well to have recourse to the following combination:

℞. Sodii salicylat.
Phenozon . . . . āā gr. v.
Syr. zingiber . . . ʒj.
Aq. chlorof. . . ad. ʒss.

M., sig.: Every quarter of an hour until pain ceases. Not more than four doses to be taken.

This is a most admirable combination in the migrainoid neuralgic attacks to which the gouty are peculiarly prone. I have appealed to it in a very large number of cases and, so far, never in vain. For a reason which I do not pretend to be able to explain the combination of these two drugs is infinitely more effectual than either given alone. When directed to be taken as above the combination acts better than in single large doses.

A fruitful and easily overlooked cause of neuralgia, especially in women, is the toxic blood state induced by chronic constipation. This must be treated by the curing of this vicious habit on the lines already laid down,* but here, again, we may have to treat the neuralgia concurrently with the treatment of the constipation, in which case the phenozone and salicylate mixture just described will usually be found the most useful, though quinine and gelsemium are to be preferred where, in consequence of the long duration of the constipation, the patient is anæmic and emaciated.

Neuralgia is considered by some as an hysterical manifestation. This, of course, it may be, though hyperæsthesia is less characteristic of hysteria than anæsthesia. Neuralgia certainly occurs frequently in neurotic women, but I have generally found that there is some discoverable and removable cause for the neurosis of which the neuralgia is an occasional manifestation, and this cause is more often than not a peripheral irritation of which the origin is to be found in the teeth, the ears, or the eyes. Such irritation, when acute, is a recognised cause of neuralgia in those who are not neurotic, and there is no difficulty in supposing that when chronic it may give rise to that nervous instability to which the term "neurotic" is applied. A common site for such irritation is the ovarian region. Examination of this region in neurotic women will often reveal pain on pressure over one or both ovaries, in which case infinitely the best drug to use is belladonna. It is usefully combined with phenozone, and its administration should always be accompanied by the application of blisters, small in size, but frequently repeated in the region where the pain has been elicited. A practical point of considerable importance in connection with the management of these cases is characteristically expressed by Goodhart in that altogether admirable little work 'Common Neuroses' in the following passage: "I have said it is a bad day for a man when he first knows he has a heart; it is a ten times worse day for a woman when the pelvic pains

* "Goutiness," Clinical Journal, January 31st, February 7th and 14th, 1906.

* *Vide* Clinical Journal, August 23rd, 1905.

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to which so many are subject are focussed for her by medical opinion upon uterus or ovary. If there is anything which curdles my blood it is to hear a woman talk of her ovaries as she might of some intimate acquaintance." In the manufacture of incorrigible neurotics the word "ovarian," blurted inadvertently from incautious lips, is a common and potent ingredient.

Irritation within the buccal cavity is a frequent cause of facial neuralgia, and it should be remembered that it is not only teeth which are obviously carious which may produce this result, but, as Savill * points out, careful examination, by tapping the teeth, or by the application of hot and cold liquids alternately, will often reveal the existence of an irritated pulp in an apparently sound tooth, the proper treatment of which is necessary to a cure. For the relief of neuralgia of facial or cranial distribution arising from such a cause, butyl-chloral is probably the best of all internal remedies. It should be given in pill form, 5 gr. every half-hour until pain ceases, not more than six pills to be thus taken. It is usefully combined with gelsemine, which is another drug with a selective influence over cranial neuralgias. For this purpose the Pil. butyl-chloral c̄ gelsemina (Martindale) is very useful. Local applications are not usually accounted of much value in facial neuralgia, but I have found that the Lin. aconite carefully painted with a camel-hair brush over the area occupied by the pain is not only helpful in assisting the action of drugs taken internally, but that it is in some cases sufficient of itself to cut short an attack. For the vague, ill-defined, neuralgic, and "rheumaticky" pains of which people not infrequently complain I have found chloride of ammonium, gr. 20, combined with Tr. cimcifugæ, ℥ 20, more effectual than any of the above-mentioned remedies.

One of the most valuable drugs for the relief of neuralgic and neuritic pains, wherever situated, is acetanilid (antifebrin). In spite of its undoubted powers in this direction, even where such pains as those of tabes are concerned, it has of late fallen into disuse. This has been due in a large measure to the fact that it is credited with the production of untoward effects. This is in reality only partly true. If the drug is properly employed it is no more dangerous than any of the numerous drugs which daily flow freely and callously, so to speak, from the point of the prescriber's pen. First, then, as to dose. The official dose of 10 gr. is too high, at any rate to start with. It is better to begin with 2 gr., which will in many cases be found sufficient. If not, the dose may be gradually increased to 10 or even 15 gr. It should not be increased above the latter if, the physiological effect having been produced, the pain fails to yield. This physiological effect is slight cyanosis. The lips and nails become a dusky red. This effect has in a great measure been responsible for the disuse into which the drug has fallen. There is nothing alarming about it, and it is necessary to remember that, as in the case of so many other drugs, the curative virtues of acetanilid very often refuse to show themselves until this physiological effect has been evoked. These facts seem worth insisting upon because, apart from morphia, acetanilid is, in my judgment, by far the most powerful anti-neuralgic at our disposal. It will relieve the pains of locomotor ataxy and of other organic diseases of the nervous system when nothing else will, and he who allows himself to be frightened by the occasional cyanosis which it causes deprives himself of a most valuable therapeutic agent. That its use requires a certain amount of caution is not a sufficient reason for abandoning it. Acetanilid, being practically insoluble in water, is best given in cachet combined either with salicylate of sodium (gr. 10) or camphor monobromat. (gr. 6).

It is scarcely necessary to mention morphia as an anti-neuralgic except to say that it is not employed as often for this purpose as it might be. It is, of course, most undesirable that its use should be prolonged, but there is no objection to exhibiting it for the relief of pain pending the action of other remedies. For the intense neuralgia which sometimes accompanies true influenza and other acute toxæmias it has no equal. Its use in recurrent neuralgias is undesirable, not only on account of the patient, but also for the sake of the practitioner, who, unless he is careful, will find himself urgently summoned at all hours of the day and night to administer the necessary dose. This, which is unsatisfactory enough, is on the whole preferable to entrusting, as is too often done, the patient with a needle and a bottle of tabloids.

Another unduly neglected means of relieving neuralgic pains, in suitable cases, is leeching. One would not, of course, recommend it in weakly,

* 'A System of Clinical Medicine,' vol. ii.

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anæmic subjects, but in full-blooded individuals the relief it gives often approaches the miraculous. This is especially true where the pain seems to be in, or to radiate from, the ear. A leech placed behind the ear and allowed to take its full quantity of blood will often give complete relief where other means have failed.

There remains to be considered a highly important, perhaps the most important of all peripheral irritants in the causation of neuralgia, which I have left to this stage that I might discuss it in association with headache, to which it also gives rise with great frequency, namely eye-strain. Where eye-strain is concerned, neuralgia and headache may be regarded as synonymous terms, for it is impossible to be certain where the one ends and the other begins. And I may say at once that these two conditions by no means exhaust the troubles to which eye-strain may give rise. It is, as we shall see presently, a frequent, though too often unsuspected, cause of neurasthenia, melancholia, intemperance, and drug habits, to say nothing of such minor matters as irritability of temper, dyspepsia, constipation, and "sluggish liver."

The first point to remember in connection with eye-strain—and it is one upon which it seems very necessary to insist—is that the condition is produced, not by gross defects, but by slight ones; not by high degrees of errors of refraction, but by minor ones.* So much is this the case that patients are often indignant that any aspersions should be cast upon their eyesight, which, they will protest, has always been exceptionally good. And the truth is that such patients *are* able to see as well as anyone, for the reason that, the defect being slight, it has always been well within their power, by contracting the ciliary muscle, to overcome the defect. In the case of those with gross defects no amount of contraction enables them to see clearly, and so the effort, even if it is ever made, is very early abandoned. In the case of those with minor defects the difficulty arises from the fact that it is no more possible to keep the ciliary muscle contracted for hours on end with impunity than it is to keep any other muscle in the body so contracted without fatigue. Especially does this apply when astigmatism is present, as the ciliary muscle is then contracted, not only continuously, but also irregularly. The difference between the ciliary and other muscles is that in the latter the symptoms of fatigue are easily recognisable and are at once referred to the seat of origin, whereas in the case of the ciliary muscle these symptoms are not easily recognisable, and so long as the vision remains unimpaired the seat of origin is almost certain to go undetected. The patient sees well, but in the majority of cases he does so at a cost which, physiologically speaking, he cannot afford to pay. He lives well up to the limit of his nervous income, and any slight unexpected expenditure in other directions, such as anxiety or a dyspeptic attack, will very readily project him into bankruptcy. It is when he has reached this state that he appeals to his doctor to be relieved of a headache or an attack of neuralgia. For, be it remembered, the pains which are caused by eye-strain are by no means always present; frequently, indeed almost invariably, they require some extraneous cause to provoke them. As long as the patient is permitted to pursue the even tenour of his way, he is able to live within his income and keep his enemy at bay, but no sooner is that even tenour disturbed, as by worry or an over-generous dinner, than he finds himself at the end of his resources, with his enemy at his throat. The factor which in a very large number of cases supplies the disturbing element is the advance of time. As long as a man is young and vigorous his nervous income is equal to almost any strain; but as years wear on this income gradually diminishes, and as he approaches the presbyopic age, when another disturbing element awaits him, he is always more or less on the margin of symptoms. When the presbyopic age is actually reached and the lens has lost a great deal of its original elasticity, the ciliary muscle has still more work thrown upon it, and consequently such symptoms are liable to be of very frequent occurrence.

The nature of these symptoms varies within very wide limits. Neuralgia and headache, with which we are for the moment especially concerned, take the first place. Neither the distribution of the neuralgia (except that it is generally cranial) nor the type of the headache afford any indication that it is the eyes which are at fault, so that it is all the more important to keep constantly reminding ourselves of the now well-established fact that where either of these symptoms cannot be traced to any obvious cause eye-strain is, in all probability,

* See 'Refraction of the Eye,' by Ernest Clarke (Baillière, Tindall and Cox).

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the main factor in their production. Another common effect of refractive errors is giddiness. This is liable to occasion very considerable alarm to patients, who are generally afraid that it indicates intra-cranial mischief. To medical men it generally suggests gastric disorders, aural troubles, or circulatory disturbances. These are all common causes, but probably the most common especially in people about middle life, is that which is least frequently remembered, namely ocular defects which are slight and therefore unsuspected by the patient.

Nausea and vomiting are often due to eye-strain. In these cases, as in most others, it is to be remembered that something more than eye-strain itself is required to precipitate an attack. That something is often so slight a matter that it altogether escapes notice unless the presence of eye-strain, acting as a chronic underlying irritant, is realised. In all these cases it is the summation of slight stimuli which produces the effect, and of these slight stimuli that which is constant and ever present is the one whose removal is important. With that gone the others cease to be operative.

But the manifestations of eye-strain are not all as definite as the foregoing.* The condition, indeed, expresses itself even more frequently in forms which, until we are familiar with its almost unlimited capacity for producing indefinite symptoms, are very difficult of recognition. 'The Autocrat at the Breaklast Table' says that "it is better to lose a pint of blood from your veins than to have a nerve tapped." Now, that is precisely what eye-strain does. It taps a nerve; the energy runs to waste and the whole cerebro-spinal system becomes exhausted. When once the outline of this picture is clearly discerned, it is by no means difficult to fill in the detail. For cerebro-spinal exhaustion, though it has no symptoms by which it may with certainty be recognised by the doctor, has a very real existence for the patient. In its slighter degrees it may mean no more than the deprivation from a particular viscus, say the stomach, of its fair share of nervous energy, leading to dyspepsia; or it may spell an evident want of control in the higher cerebral centres, causing irritability of temper, undue emotionalism, or a craving for stimulants. In more pronounced degrees it will cause the grouping of symptoms to which the term "neurasthenia" is applied; it may cause hysterical ebullitions, and may even be responsible for epileptic attacks. In degrees still more pronounced it may lead to melancholia and even to suicide. There is, in short, no functional disturbance of any portion of the central nervous system which may not own eye-strain as its essential cause, so that it is impossible to insist too strongly upon the importance to every practitioner of being able to detect slight errors of refraction when they exist. Into the details connected with the necessary examination it is impossible to enter here, but the following hints may serve as a useful guide.

* See "The Medical Aspect of Eye-strain," by Ernest Clarke, CLINICAL JOURNAL, October, 1905.

(1) *Objective Examination.*

(*a*) Defects of vision may be suspected if the patient screws up his eyes or places his head on one side in order to read or to see some object at a distance. If there is a hyperæmia of the margin of the lids, generally the upper lid, an error of some kind is usually the cause, and if a patient under forty years of age presents an arcus senilis, especially if one eye only be thus affected, it is almost certain that the premature degeneration has been brought about by eye-strain of some kind.

(*b*) If the eye is examined by the *indirect* method with a concave mirror and focussing glass:

Hyperopia is present if the disc is larger than usual and diminishes on withdrawing the glass from the eye.

Myopia is present if the disc is smaller than usual, and enlarges on withdrawing the glass, and

Astigmatism is present if the disc is oblong and alters in shape on withdrawing the focussing glass.

(*c*) By *direct* ophthalmoscopy.

Hyperopia is present if convex glasses improve the view of the fundus, *myopia* if concave glasses do so, and *astigmatism* shows itself by parts of the fundus being out of focus while other portions at right angles are in focus.

(2) *Subjective Examination.*

Hyperopia is probably present if the patient's vision is not made worse by convex glasses.

Myopia is suspected if the patient's reading distance is nearer than normal, and

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Astigmatism if the patient can read some of the letters in the lowest line of the distant type but makes mistakes even when reading a line half-way down, or if when looking at radiating lines put at a distance of 4-6 metres, some of the lines look blacker than others.

It must, however, be distinctly borne in mind that these tests, if *negative*, prove nothing if the patient is not under the influence of a cycloplegic, as he may be, and probably is, involuntarily correcting his error.

Eye-strain is, however, by no means the only kind of chronic peripheral irritation which may give rise to headache. Dental troubles which fall short of gross caries frequently produce it, overcrowding of the mouth owing to eruption of the wisdom teeth being a common and often unsuspected cause. Impairment of respiratory power, brought about by obstructions in the nose and throat, are other easily overlooked causes, chief among them being deviations of the septum and other factors acting upon one nostril only. Aural troubles of every sort should always be carefully examined for.

A form of headache which presents special features and concerning whose etiology there has been a great deal of speculation is migraine or hemicrania. The latter name is applied to it because the pain generally begins on one side of the head, and is sometimes confined to that side throughout the attack. The characteristic of migraine is its periodicity. The attacks recur at intervals, though not necessarily at regular intervals, and their origin is exceedingly difficult to trace. Migrainous people, like epileptics, are always more or less liable to an attack, and, like epileptics, they have premonitions; they know the sort of influence which may affect them, and immediately after an attack they know that they will enjoy immunity for a variable time. The attacks usually begin, significantly enough, with some ocular phenomenon, such as flickering lights of various forms and zigzag lines of colours in different parts of the field of vision. Then follows an intense headache, accompanied generally by nausea, vomiting, and extreme prostration, which may last for varying periods. At the end of forty-eight hours the patient is generally well again. To understand the phenomena presented by migraine we have, as in epilepsy, to assume some underlying instability of the nerve centres, which are provoked into a stormy condition by stimuli which pass by ordinary individuals without effect. The attacks are common in the studious, in the sedentary, in the highly-cultured, and are rare in the bucolic. In the light of what has been said above on the question of eye-strain and the capacity of this condition to produce a disturbance of nervous equilibrium, coupled with the fact that migrainous attacks are almost invariably accompanied by ocular phenomena, very especial care should be taken to eliminate this element in every case. A very large number of people have slight defects of vision, and such slight defects, while harmless to the peasant of robust organisation, may very readily provide a constant irritant to the studious of delicate organisation, and thus contribute a powerful underlying cause for the attacks. A migrainous person should be examined very minutely by physician, oculist, aurist, and gynæcologist, and any defect in any department, however slight, which could possibly act as a cause of irritation, should be removed. The physician should pay particular attention to the cardio-vascular system, the oculist to the state of the refraction, and the aurist to any remediable errors either in the ear, throat, or nose. If nothing can be discovered, then we are reduced to attempting to lessen the general nervous reactive sensibility by hygiene, diet, and the exhibition of bromides. A country, open-air life, a diet free from stimulating foods and alcoholic drinks, and the bromide of ammonium in 10-gr. doses, three times daily, for a week every now and again, will very often keep the enemy at bay. During the attacks rest and a darkened room are essential. When taken in time—that is, before the attack has had time to develop—the phenozone and salicylate mixture above prescribed will often, indeed generally, act admirably. I have found it superior to phenacetin (gr. x), in which some people, however, express great confidence.

Upon headache as a symptom of intra-cranial mischief it is unnecessary to dwell. When the pain, as in migrane, is accompanied by vomiting, the fundus oculi should always be examined for anything suggestive of optic neuritus, and careful search should be made for other physical signs of organic disease in the nervous system. Intra-cranial tumours and meningitis are causes which should always be borne in mind.

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There is a certain class of headache which is occasionally described as congestive. The term is misleading because headaches due to a diversity of causes might legitimately be so called. If, however, we qualify the term by the adjective "mechanical," it is possible to recognise a separate and distinct group. Headaches due to mechanical congestion are produced by factors which are easily overlooked. Anything which interferes with the proper circulation of the blood may occasion them, and in ordinary life tight corsets in women and tight collars in men will be found to be common causes. This type of headache, which is described as a general fulness, aggravated on stooping, may be the first indication of the existence of a lesion at the mitral valve, so that a complaint of such nature, more especially when it is accompanied by a history of epistaxis, should always lead to a careful examination of the cardiac area. Nasal obstruction, whether from enlarged tonsils, adenoids, or deviations of the septum, is exceedingly likely to cause headaches of the congestive type. The two former are nowadays very readily recognised, and very promptly—perhaps too promptly—removed, but the latter is often allowed to continue unremedied. Nasal deformities constitute an exceedingly common cause of headaches in adults, and as they are now easily remedied, there need be no hesitation in advising their radical cure, even in people who are past middle life. The correction of these deformities must be left to the expert, but I would suggest that the most speedy, complete, and lasting benefit is to be obtained from the recently introduced method of submucous resection, by which the cartilaginous and bony obstruction is entirely removed, leaving a fleshy septum, an intact mucosa, and a clear air-way.*

Headache is very often caused by change of climate. The removal of a person from sedative to bracing conditions, and even more frequently *vice versâ*, commonly brings about changes in the vascular pressure, which, in the absence of adequate reactive power in the individual (in the convalescent for example) very frequently cause headache and depression of spirits. If these symptoms do not pass off in a few days, it may be necessary to resort to treatment. Where the headache has been induced by relaxing climatic conditions a mixture containing 2 gr. of quinine and 5 min. of Liq. strychnin. will generally do all that is necessary. Where, on the other hand, the climate is "too strong," as the expression is, ten grains each of the iodide and bromide of potassium, three times a day, will be found useful. In each case the mixture should be preceded by a dose of calomel.

Pain in the head of a superficial character, such as seems confined to the scalp, is a frequent accompaniment of "colds" in the head. In such cases the pain is usually due to a fibrositis of the tissues overlying the skull, and is to be treated on lines already suggested. Headache is a common symptom of neurasthenia.

Most headaches are due to what are comprehensively described as blood-states. A good instance is supplied by chlorosis, in which the headache is due to the abnormal condition of the corpuscular elements, and the same is presumably true where the symptom appears in leukæmia and other associated conditions.

The offending blood state in the majority of cases is, however, a toxæmia. There are numerous familiar instances of this. All the acute specifics are liable to be ushered in with a headache more or less severe, and the poisons of alcohol and lead almost invariably cause the symptom. Among the toxæmias due to defective excretion, kidney disease and diabetes are classical instances of causative factors, while chronic constipation is probably both the commonest and the most generally overlooked. In all these cases the discovery of the cause immediately points the way to the proper treatment, and some people have endeavoured to aid in the discovery of the cause by an attempt to map out certain cranial areas in which pain occurs and attaching to each area a particular group of causes. Thus, pain which is mainly vertical is said to be due to the following: anæmia, hysteria, neurasthenia, epilepsy, and disease of the uterus and appendages. A headache which is chiefly frontal is deemed gastro-intestinal, renal, ocular, syphilitic, or myalgic. An occipital headache is considered suggestive of intra-cranial mischief and spinal irritation. While believing a great many of these to be fanciful, I think there is no doubt that headaches due to defective elimination, such as those caused by uræmia, diabetes, constipation, and

* StClair Thomson, 'Proc. Laryngol. Soc. Lond.,' May and June, 1904; 'Trans. Clin. Soc. Lond.,' October 25th, 1905; 'Lancet,' June 30th, 1906.

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impure atmospheres are generally frontal, and that those which appear in functional nervous troubles seem to have a preference for the vertex, but even in such cases it is not wise to attach much importance to so variable an element as the site.

In everyday life it is certainly the toxins of gastro-intestinal origin which are most frequently responsible for the occurrence of headache. These toxins most frequently act by interfering with the intra-cranial vasomotor mechanism, causing now undue contraction and anon undue dilatation of the cerebral vessels. The indications in either case are the same, namely to cut off the supply of the toxins and to promote the excretion of those already in existence. The means of doing this I have already discussed* so that here it is only necessary to emphasise the importance of dietetic simplicity, such as absence of meat foods and alcoholic drinks, and of excretory vigour by means of purgatives and diuretics. Stress may perhaps be laid on the importance of the latter, which seem very generally to be neglected. The salts of potassium, especially the iodide and the citrate, are most useful, but the best of all renal stimulants is undoubtedly theobromine. Some physicians who are possessed by the uric acid bogey, object to this drug on account of its close relationship with xanthine and the other purin bodies. An over-curious regard for these would, however, lead also to the condemnation of caffeine, whose efficacy as a heart tonic in suitable cases is second only to that of digitalis. Whatever its connection with other bodies with the radical C_5N_4, and however dangerous such a connection may prove it theoretically to be, there can be no doubt that, clinically, theobromine is the most active renal evacuant we possess, and it may be prescribed with every confidence in all cases where we are desirous of ridding the system of a toxin, such as a nitrogenous toxin, which is normally disposed of through the kidneys. It is best given in cachets of xv–xx gr. three times daily. It occasionally operates as a drastic purgative, in which case the dose must be lessened. Some people prefer to give it in the form of diuretin, in which it is combined with a salicylate.

In addition to general evacuants it is generally desirable to prescribe measures directed to the relief of symptoms. Where there is reason to suppose that the headache is due to vaso-constriction, Liq. trinitrin is very useful. Its effect, however, is transitory, and it should not for obvious reasons be frequently repeated. It is best given in conjunction with hydrobromic acid, thus:

℞. Liq. trinitrin . . . ♏iv.
Acid hydrobrom. dil. . . ♏xx.
Aquam . . . ad. ℥ss.
M., sig.: Ter die.

Another useful drug for the symptomatic treatment of headache is Cannabis indica. If given as the tincture in combination with other drugs it must be suspended in mucilage. It is, therefore, better to give it separately in the form of extract, of which half a grain to a grain may be made into a pill with lycopodium, and given three times daily.

Ross, in a valuable paper contributed to the 'Lancet' of January 20th, 1904, describes a chronic headache, which he calls the lymphatic type of headache, which shows itself as a dull, heavy ache, worse in the morning, and tending to wear off as the day advances, and is accompanied by mental and physical lassitude. This, he says, is associated with deficient coagulability of the blood, and yields readily to chloride of calcium.

In very persistent headaches which resist all treatment it is well to try the effect of a blister in the nape of the neck to be kept open with savin ointment for a week or ten days on end. I have known this expedient succeed where other measures had failed.

August 20th, 1906.

* "Goutiness," CLINICAL JOURNAL, 1906.

Differentiation of Pleural Effusions from Pneumonia.—Krönig has found a triangular area of resonance in cases of pleural effusion, which is contrary to the teachings of the text-books. His experience with nearly a thousand cases has convinced him that this area may serve to differentiate a pleural effusion from pneumonia in dubious cases. This area, comparatively free from dulness, lies between the effusion and the spine, and testifies to the fact that at this point there is little, if any, fluid. Krönig found the highest point of the area of resonance at the inner margin of the scapula, the upper limit sloping gently from this point toward the axilla and the spine.—*Journ. A. M. A.*, vol. xlvii, No. 3.

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INTERNATIONAL CLINICS, vols. iii and iv, Fifteenth Series. J. B. Lippincott Company, Philad. and London, 1905.

The place of honour in the first of these volumes is given to a paper by Dr. George C. Johnston on the therapeutic uses of radiography. The technique is described and then the various forms of disease suitable for this form of treatment. The paper is very fully illustrated, and apparently many of the cases are cured, although the appearance of the patient when that result has been achieved is not always all that could be desired. The subject is one which can be treated only by specialists, and even they have not yet had time to draw final conclusions. As Dr. Johnston remarks: "The field of usefulness of this agent will broaden steadily with the lapse of time, and the success or failure of the individual operator will be credited, as they should be, not to the agent employed, but to the skill or lack of skill with which he employs it." The second volume contains an interesting article by Dr. A. S. Warthin on the effects of Rontgen rays on the blood-forming organs, and more especially their therapeutic value in leukæmia. His experiments have confirmed the results obtained by Heinecke, who found that the rays had a selective action on lymphoid, myeloid, and epithelioid cells, causing nuclear disintegration and necrosis. By prolonged exposure practically all the lymphoid tissue of the spleen may be destroyed. In the treatment of leukæmia the improvement noted is due to the removal of leucocytes from the general circulation, and from the spleen and other organs where they collect, and to a delayed production of new cells. But, *per contra*, the destruction of enormous numbers of leucocytes in the circulation by means of the rays may in itself be dangerous, and from the disintegrating cells poisons may be formed which may cause a fatal intoxication. He concludes that the therapeutic use of the rays in leukæmia seems of doubtful value, and may be dangerous. Dr. Huchard, of Paris, has an interesting article on the musculo-tonic and diuretic action of formic acid. This is based on the work of Dr. Clement, of Lyons, who has been recording wonderful results from the use of the formiates. The painful sensations caused in muscles by repeated contractions are noticeably lessened, while the tired muscles quickly recover their energy. The musculo-tonic action seems to affect all the muscles, striated and unstriated, of the body. The circulation is improved, the respiratory muscles are strengthened, and visible awakening of appetite and active digestion are obtained. Formic acid seems to be *a real elixir vitæ*. Dr. Huchard has not yet had experience of the drug for a sufficiently long time to be able to support all these claims, but he found by experiment that formiate of soda had the effect of raising his muscular strength from 8·75 kilogrammeters to 30·65, an increase of nearly four times in a space of six days. He suggests that formic acid probably explains the activity, strength, vigour, and endurance of certain working insects—caterpillars, and particularly ants—which are able to carry loads out of all proportion to their size. We shall await further information as to the therapeutics of the formiates with interest, and hope that Madrali may be induced to test a drug which might turn defeat into victory. Striking clinical results are also recorded by Dr. Trissier from the "opotherapic treatment of renal insufficiency." He describes three methods. First, a glycerine extract of a pig's kidney, named nephrine, is injected subcutaneously. Secondly, the renal venous serum of a goat is used subcutaneously. Thirdly, the macerated kidneys of sheep, pigs, and calves are given by the mouth—integral opotherapy. The clinical indications for this line of treatment are, sudden signs of renal insufficiency occurring in the course of renal disease. Of the three methods he prefers the second, but admits that at present the renal venous-blood of a goat is not always at hand. The last paper in the therapeutic section is by Dr. Wainwright, of New York, who describes the serum-therapy of various diseases. We have said enough to show that the field of medical therapeutics is occupied by many workers, and that many fresh observations will be found to stimulate thought. In a surgical paper Dr. Deaver urges strongly the calling in of the surgeon in all cases of chronic stomach trouble which have not yielded to medical treatment within a reasonable period. His great panacea for all such ailments as chronic gastric ulcer or chronic dilatation is drainage by gastro-enterostomy. Perhaps at the present time physicians are rather backward in adopting such measures in suitable cases, whereas surgeons are rather too ready to regard every case of chronic stomach trouble as amenable to surgical treatment. Time and careful clinical observation will enable the practitioner to decide how far and in what cases surgical intervention is of value. There are many other topics of general and special interest discussed in these volumes, which will well repay perusal.

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A CLINICAL LECTURE

ON

PYURIA.

Delivered at St. Bartholomew's Hospital.

By W. P. HERRINGHAM, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—There is, lying in the back ward of Mary, a thin, dark, rather delicate but not unhealthy-looking girl, who is passing the urine which you see in that glass. You notice that it has been recently passed, so that the deposit has not yet settled. But it is pale and rather turbid urine, with a white deposit at the bottom which when finished will be an inch to one and a half inches deep. Now, that girl is an old patient of ours. She came under me at the end of August, 1904, and the history that she gave then was that for three months she had noticed that her urine was turbid, that for four months she had been losing flesh, that her appetite was less, and that a fortnight before she came to us, when she was at Ramsgate, she had bathed in the sea, and the same night noticed a severe pain in her left side. This became so bad that she had to go to bed. She had been in bed practically the whole fortnight before coming to us. The urine then was exactly like this, and under the microscope we found that there were pus-cells. She had pyuria.

You cannot always find pus-cells under the microscope. When the urine is acid or very faintly alkaline, as hers always was, or neutral, the leucocytes are well preserved, and it is easy to find them; but when you get urine strongly alkaline or ammoniacal the cells are broken up and dissolved, and you cannot find them under the microscope. That is the case with this other urine here, which, again, is freshly passed, so that it is not a very good specimen for our purposes. It is higher coloured than the girl's, but it is turbid, and when it has finished depositing there will be a

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white precipitate like this other. It is from a case of myelitis, the one which I lectured to you about at the beginning of last term. If you look at the thick deposit which will come down shortly, under the microscope, you will find that a great part of it is formed of crystals of ammonio-magnesium phosphate, and others. There are also a few cells half dissolved, with edges broken and irregular, but the rest of it is an immense mass of granular material, the cells broken up by the ammoniacal condition of the urine. When you cannot see pus under the microscope you are driven to have recourse to such chemical tests as you can use, and none of them are good. One test which is highly recommended is the addition of tincture of guaiacum, which will leave a blue colour with some specimens. I have tried that with the first urine, where there is plainly pus, and it does not give the reaction, although the reaction is said to be a good one. Another is the addition of Liquor potassæ, half as much Liquor potassæ as there is urine. That makes the urine into a mass like a piece of swollen gum, like what you get if you add water to mucilage. That is about the best chemical test we have for pus in urine, and you will see how poor a one it is. Another consists in the addition of ozonic ether. When it is shaken up it gives off oxygen and you get a solid mass at the bottom, with a lot of little bubbles coming off. Both those tests are exceedingly imperfect, and there is no rhyme or reason in them. Although I know that this man is a case of purulent cystitis, because we watched it from the first when there were crowds of pus-cells, yet if we were to see the urine now for the first time we should have a difficulty in being sure that it was purulent.

When you have recognised that urine does contain pus, the next question is, where does the pus come from? For, just as in the case of blood in hæmaturia, pus may come from any part of the urinary tract. It may come from the urethra; that is just the ordinary case of gonorrhœa. When it is from the urethra it is passed just before the urine is passed if the patient makes water, and you can also squeeze the pus out of the meatus without the patient passing his water at all. It may also come from a collection of pus in the prostate. Some years ago a poor clinical clerk of mine came to me and said he was passing turbid urine. I found that his urine was loaded with pus, and it was due to a tubercular focus in his prostate. This constituted the beginning of genito-urinary tuberculosis, from which, I am sorry to say, he died. More often it comes from the bladder, and on these occasions it is very common to find the urine alkaline, like the exudation which comes from any mucous membrane when it is inflamed. And very often it is ammoniacal, from the microbic decomposition of the urea contained in the urine. Now, the cystitis of which that is a symptom may be due to many causes. In the first place it may be traumatic, as from a calculus; or in the second place it may be septic, as, for instance, in this case of myelitis; and in the third place it may be due, not to general septic organisms, but to a specific infection—for instance, after typhoid. We frequently see cystitis due to the typhoid bacillus or to the tubercle bacillus, in which latter case it is commonly associated with ulceration.

But I have had another case recently in the medical wards which is so rare that it completely misled me for a little while. It occurs so uncommonly in medical practice that I will tell you about it. It was a man, æt. 24 years, who came in with the story that for three weeks he had had pain at the end of his penis. For a few days he had hæmaturia, and for about three weeks he had great irritation of the bladder, so that he was constantly having to pass water. When he came in we found that his story was true. He had to micturate every quarter of an hour. He was passing blood with his urine, and when it was placed under the microscope we found a good deal of pus. We diagnosed cystitis without hesitation. And the question was whether it was calculous, which seemed to be likely from the history, or tubercular. But it was not until ten days after he came in that he began to pass pus from his urethra. He certainly had no urethral discharge when he came in. This pus contained the gonococcus. We took the proper steps to cure his gonorrhœa and the urethritis due to it, and as that cleared up so also did the cystitis. I have no doubt the cystitis was gonorrhœal, and owing to the delay in the suppuration appearing from the urethra we were entirely misled in the diagnosis. We found afterwards that he had an old-standing gonorrhœa, which had subsided, but which had probably been awakened freshly in his bladder, and did not appear in his urethra until later.

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If pyuria is not due to trouble in the bladder, it is probably due to the kidneys, either from the pelvis of the kidney or from the body of it. But it can hardly affect the body of the kidney without affecting the pelvis, because it has to go through the pelvis, and the same infection will attack that as the body. In those cases the urine is often acid, though sometimes it is neutral or very faintly alkaline. That is, and has always been, the case with this girl. Such cases of pyelitis, or pyelonephritis, may be septic. Septic pyelonephritis is generally ascending. It forms the great danger in cases of myelitis. This man whose urine I have shown you to-day will, I hope, recover without any such extension of his cystic infection. But some years ago we had, in Colston ward, a young fellow with very much the same complaint. He was a ship's engineer, and had been taken suddenly ill with paraplegia. He had been in hospital in Trieste for nine months, and had partially recovered the use of his legs and sensation, but he was left with a very bad cystitis and pyelonephritis, from which he died. He died of his pyelonephritis, not of his paralysis. And there is another way, although it is much less common clinically, in which you may get suppuration of the kidney, and that is by a blood-borne infection—in cases, for instance, of septic endocarditis. These latter cases are of but little clinical importance, because the affection of the kidneys is usually quite slight. But both kinds are acute and fatal, the first because of the great severity of the local disease and the second because of its even greater severity as a general disease. They do not cause any difficulty in diagnosis; the cases which are really difficult to diagnose are chronic pyuria of the kidney. Those are nearly always due either to calculus or tubercle in the substance of the kidney.

Now, you can not always diagnose where the pus is coming from by mere examination of the urine. You often have to rely upon the symptoms which the patient tells you of. For instance, in the case of gonorrhœa we had not the least hesitation in saying that it came from the bladder, because his symptoms all pointed to the bladder, there was pain at the end of the penis, and continual irritation leading to frequent micturition. Similarly with this girl we had no hesitation in saying it came from the kidney; her symptoms consisted entirely of pain in the loin, and she did not complain of any frequent micturition or of any irritation in the bladder. When we came to examine her we were rather surprised to find that although all her symptoms were referred to the left side of the abdomen, she had on the right side a very large, hard mass which was obviously renal. She had a large tumour of the right kidney. There was no doubt that the right kidney was affected then. But the questions still remained, had she any infection of the left kidney? What was the nature of the disease? Now, it is very often of great importance to be able to diagnose accurately the condition of each kidney separately. You can sometimes get a very fair idea of it with the cystoscope. By putting that instrument into the bladder you can see whether from one ureter there is a perfectly clear secretion and diseased secretion from the other, or whether the mouth of one ureter is healthy and the other one obviously diseased. That gives you a very good idea of whether one kidney is fairly sound and the other diseased. But no really satisfactory idea can be obtained of the condition of the two kidneys except by collecting and analysing the secretions from them separately. And you can do that in one or two ways. One way is by introducing a separator into the bladder. Here is the instrument, and you see the principle is simply that you put it into the bladder, and by turning this handle you bring into the middle an india-rubber division, so that the urine which falls from this ureter passes down the tube on that side, and the urine from the other on this, and it is collected separately into two little glasses. That instrument is fairly satisfactory in the case of women, but it is painful to use it in men. Such a large curve is exceedingly difficult to introduce, and it cannot be borne by most men without an anæsthetic. In women it is tolerated, and we used it in our girl patient several times. Of course, it is liable to the objection that if you do not hold it very carefully the urines may mix underneath here, and you can only be sure that that is not the case by finding that the secretion from the two sides is very different. We examined her several times in that way, and the results were very informing. They were very different in quantity, though not so different in the percentage secretion of the solids. The last separation gave practically this: that in one hour the right kidney secreted 120 cm. and the left only 30, that the urine from the right

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kidney was very pale and limpid, and with a specific gravity of only 1002, so that it was little more than water. The urine from the left was thick and turbid. The right kidney secreted in an hour one gram of urea, while the left secreted only one fifth of a gram. The secretion of chlorine was not quite in the same proportion: it was about three times as much on the right as on the left. In passing I may remind you that kidneys, especially those which are diseased, are not always permeable in the same proportion to different substances. The right kidney secreted five times as much urea as the left, but only three times as much chlorine. That estimation was only one of several, but all pointed to the same proportions. Evidently the girl's left kidney was diseased, and the other was doing nearly the whole work of her organism. The second method is by passing a catheter into each ureter; it is not often used in England.

Then came the question, what was the nature of the disease? Both kidneys were affected, but the right was the more useful of the two. What was the nature of the disease? The look of the girl made me very much inclined to regard the affection as tuberculous, and I think that would be your impression if you saw her. Probably many of you have seen her already. She is very dark, and a thin, delicate-looking girl. Of course we examined her carefully to see if we could detect tubercle in any other form, but we were unable to do so. It was necessary to examine the deposit in her urine for tubercle, and we did so. To examine the sediment of urine for tubercle bacilli as it stands is practically useless; you can hardly ever find them under such circumstances. It is much more easy if you take the precipitate and centrifugalise and wash it, and repeat that process three or four times before staining. Another method is to digest it for twenty-four to forty-eight hours with a little pepsine. Either of these methods makes the recognition of tubercle bacilli easier in the deposit. However, we could find none, although we examined it several times. So we had recourse to inoculation of a guinea-pig with it. If there are any tubercle bacilli that is usually, though not always, successful. Though we did three inoculations into guinea-pigs with this material, not one of them had tuberculosis. So we were driven to the conclusion that she had not tuberculosis of the kidney, and that therefore she had the other condition which could give her chronic suppurative pyelitis, namely stone, and that she must have stone in both kidneys. That was borne out by the results of Roentgen-ray photographs.

This skiagram shows, even at a distance, a mass on the right much the shape of a kidney turned the wrong way the hilum being outwards, and on the left a smaller mass, oval in shape, with a truncated apex at the top. That is so like a picture of calculus in Walsham and Spencer's 'Surgery' that I have brought that book for you to compare the two. That was done eighteen months ago. We had the same thing done again and it is not altered a bit. And the fact that there has been no alteration in eighteen months I think renders it practically certain that it must be calculus and not a focus of tubercular suppuration, which you may know will also give shadows. We therefore had to consider that she had a couple of stones, one in each kidney, and that she had suppurative pyelitis going on, certainly in the left, and probably to a certain extent in the right also.

What were we to do? The prognosis in such a case as that cannot but be serious if the condition is left to itself. In the first place, on the right side she has a certain amount of suppuration going on in the pelvis round the stone. Judging by the urine, with its low specific gravity, 1002, she has also interstitial nephritis, due to secondary inflammation spreading from the pelvis of the kidney, as generally happens in cases of pyelitis. On the left side she has obviously very much more acute suppurative pyelitis, and she has probably, although one cannot say for certain, some sort of secondary degeneration in her parenchyma. So one can only say that that sort of condition, if you leave it alone, is bound to progress, and ultimately will come to a fatal termination, though not nearly so quickly as you might think. In 1903 we had a woman in Mary Ward, about thirty-four years of age, who had very severe pyuria, and she had had it for four years then. By the aid of the speculum the mouths of both her ureters in the bladder were examined. Both were secreting pus, so that we knew the pyuria was double. I had not the slightest doubt then—and I have very little now—that the case was tubercular in origin, but she is still alive (I had a letter from her only yesterday), so that for nearly seven years she has been going on with double

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pyuria. And although she is not feeling so well now, she is obviously not yet in a dangerous condition. So you see if you leave even double pyuria alone it may run for several years. And if that is so in a tubercular case, it is likely that a simple one, one which is due to stone, will last longer. On the other hand, if you interfere with this case by operation, either nephrotomy or nephrectomy, the probability is that you would kill her within a few weeks. It is not so much because of the danger to the girl in her present condition of health, but that if you operate upon one kidney when the other is diseased you are very apt to cause reflex inhibition of secretion, and, in fact, acute suppresion of urine, with a naturally fatal result. So much is that the case that a surgeon will hardly ever interfere with a case of that sort except as a last resort. We consulted Mr. D'Arcy Power in the matter, and he fully took our view. So we have been driven to take refuge in palliative measures. She comes up every month, and we take her into the hospital when she has a bad attack of pain, as she had a week or two ago. Between those attacks she seems to get about pretty well, and is able to do her work as a machinist. She takes tonics and cod-liver oil, and acid phosphates of sodium to acidify the urine. Also she took urinary antiseptics, such as urotropin, which at one time produced good results. The patient is now four pounds heavier than some months ago, so I do not think there is any present danger of a fatal result.

August 27th, 1906.

As the result of investigations extending over a period of many years, with the aid of expert assistance, Sir Alnroth E. Wright, M.D., F.R.S., has written a book which Messrs. Archibald Constable will shortly publish, which cannot fail to have a marked effect on the results obtainable by all students using the microscope; hitherto this instrument has been all too commonly treated by rule of thumb, and without those considerations of the principles of microscopic technique which are so essential to the attainment of accurate and scientific results. The object of the book is to show how best to get from any class of instrument the highest degree of satisfaction in proportion to its range. The text of the whole book goes hand in hand with experiments. The work also contains a complete vocabulary of technical terms relating to the microscope.

SOME REMARKS
ON
DEFORMITIES OF THE ARCH OF THE FOOT AND OF THE TOES.*

By EDRED M. CORNER, M.A., M.B., B.C.Cantab., B.Sc.Lond., F.R.C.S.Eng.
Surgeon to Out Patients, St. Thomas's Hospital; Senior Assistant Surgeon to the Children's Hospital, Great Ormond Street.

Deformities of the Arch of the Foot.

In the conclusion of an essay Mr. Arbuthnot Lane gave this advice: "Do not lose sight of general principles, and do not let details of trifling importance occupy your mental vision to the exclusion of the former, which are vastly more important, and to which the latter, though deserving thoughtful attention, bear but a very subsidary relationship." This dictum has led me to the publication of some notes made five years ago on the natural history or associations of the deformities of the feet, because only by means of such a general study is it possible to survey a deformity in all its bearings and without such an estimate all treatment must be inadequate. Before proceeding, it may be premised at once that stress is not laid upon deformities which are the result of paralyses, but rather upon the so-called acquired deformities. Such deformities fall into three heads—those at the ankle-joint, the mid-tarsal joint and the metatarso-phalangeal joints. In the first class are the deformities of equinus and calcaneus, in the second pes planus and pes cavus, in the third deformities of the toes. The study of the natural history of the deformities of the feet has led to some classes or varieties of deformity being recognised for the first time. Further, as the characters of the deformities have been estimated from a large point of view, it has become obvious that the names in present use have been given from a narrow standpoint, and in consequence will be discarded at no very distant date. I shall use the old names, as the passage of time will bring about the changes in nomenclature more surely than if an attempt at alteration is made now.

The deformities of the foot are, unfortunately, treated in the text-books as a number of separate and distinct subjects, whilst if one studies them thought-

* Delivered at St. Thomas's Hospital.

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fully, it is found that they form a series, the members of which intermingle with each other. In this way a great branch of surgery becomes a coherent whole, whilst formerly it consisted of many isolated parts. To Mr. Arbuthnot Lane must be given the credit of being the first to organise and marshall the deformities of the foot into an orderly array. He pointed out that naturally the foot could assume two extreme physiological positions, one of which is associated with activity and one with rest. It has been argued and demonstrated that the whole of evolution bears on the physiology of the organism. All structural variations are initiated by adaptation to or for some special function. It is to physiology, not to anatomy, that we have to look for the problems of heredity, and Mr. Lane's classification, being based on physiology and not on mere anatomy, becomes of the very first importance.

There is one period of life during which acquired characters are easily impressed on the individual, namely the period of growth, when, on account of its property of growing, living matter adapts itself to the conditions under which it can exist, much in the same way as liquid gelatine adapts itself to the shape of the vessel into which it has been poured, and when it has solidified its ultimate appearance is the counterpart of the mould or vessel. Growing matter can only adapt itself to an extent limited by its capacity to exist. The lesson which this teaches is that particular care must be exercised during the period of growth to see that bad physiological habits are not allowed to be made permanent on the anatomy of the body by the agencies of growth and the adaptive lengthening or shortening of the muscles and ligaments.

The Position of Activity or Adduction.

The position of activity of the foot is one of adduction; the movement largely takes place at the mid-tarsal joint. The foot is either directed straight forward or slightly turned inward. This is the position assumed in activity, such as jumping or running, but a most important addition must be added. When running or jumping the foot is not only held in a position of adduction, a movement taking place at the mid-tarsal joint, but it is extended or, more correctly from the anatomical point of view, flexed at the ankle-joint, the individual springing from his toes with the heel raised. Thus we find the position assumed by the foot in activity to be one of flexion at the ankle-joint and adduction at the mid-tarsal joint, with an increase of the plantar arch. The movement of adduction of the foot is not so simple as it often is in other joints of the body, being accompanied by some internal rotation or inversion, corresponding directly in amount to the degree of adduction assumed. The position of the foot in activity is, in the language of deformities, therefore, one of *equinus* at the ankle-joint, *varus* at the mid-tarsal joint, and *cavus*, an increase of the arch of the foot. If for any reason or reasons the position becomes pathological, having to be regarded as a deformity, it is called *talipes equino-varus*. We

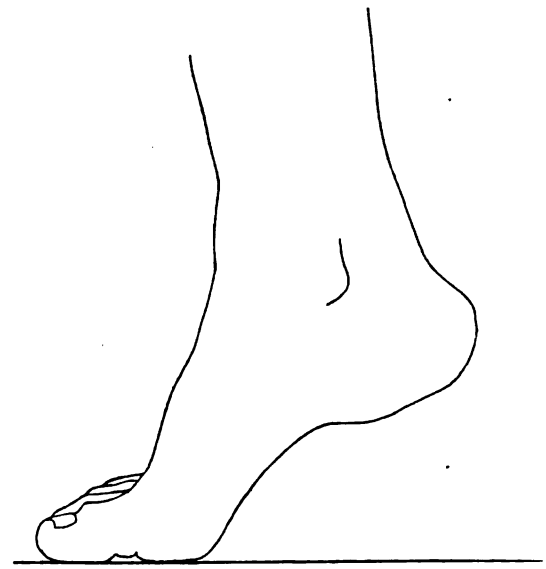

Fig. 1.—The foot in activity.

have not yet finished with the position of activity; all the toes are extended dorsally at the metatarso-phalangeal joints, flexed at the interphalangeal joints and perhaps extended at the distal joint, digiti extensi. When the position of the toes are pathological they would be called hammer-toes, From its situation, the second and longest toe suffers most and, in consequence, becomes the painful one. With the position of activity, the big toe is adducted and turned inward with the rest of the foot; but owing to the position of the foot it is not noticed, otherwise it would have received the name of *hallux varus*. The position of the big toe is not constant on account of its being displaced outward by the boot in wear.

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It is thus possible to deduce certain rules which can be confirmed by daily observation.

(1) Strong and active people are far more apt to turn their toes in.

(2) People who turn their toes in usually have high insteps—*i. e.* some degree of pes cavus—and good calf muscles.

(3) Owing to the extension of the toes at the metatarso-phalangeal joint, the points of their boots are curled up "at the toe," and the upper creased transversely opposite these joints (Fig. 2 A).

(4) The soles of their boots are worn fairly evenly and particularly at the back of the heel.

(5) The skin of the sole of their feet is worn into callosities at the tip of the heel, under the big toe, and under the third toe (Fig. 5).

(6) The position of activity is associated with a tendency to the following deformities of the foot: (1) talipes equinus, (2) talipes varus, (3) pes cavus, (4) hallux varus, (5) digiti extensi, (6) hammer-toe.

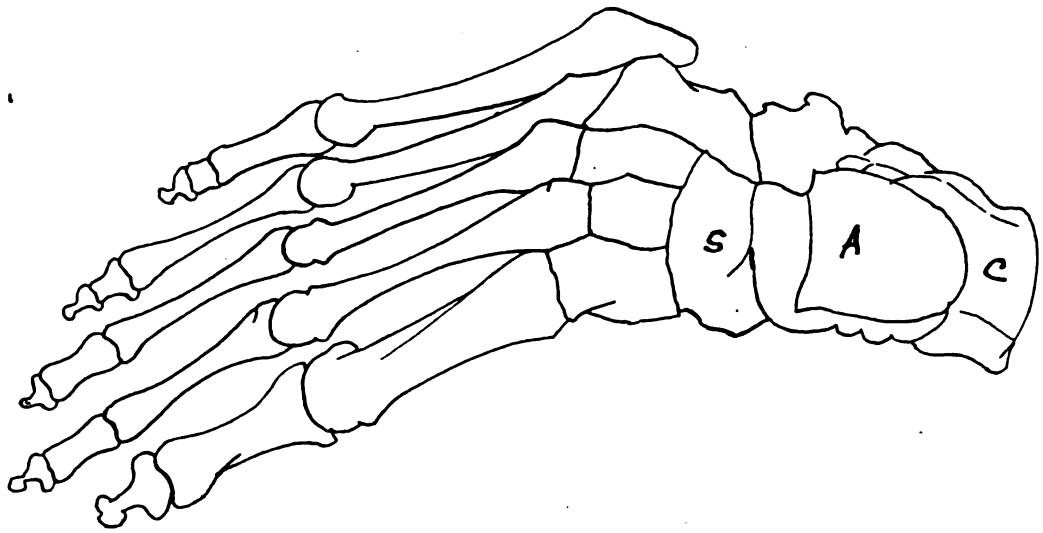

FIG. 2.—The bones of the adducted foot.

THE POSITION OF REST OR ABDUCTION.

The position which the foot takes when resting can be far more briefly described, because it is, as its name implies, precisely the opposite to the position of activity. The latter has been shown to be one of adduction at the mid-tarsal joint; the former is one of abduction at the same point. As in the case of adduction of the foot, the movement of abduction is not simple but is combined with some rotation, in the latter case with eversion, whilst in the former it was with inversion—that is to say, for rest and abduction, the valgus position. The position of rest, like that of activity, is also accompanied by a movement of the ankle; in activity it was flexion; in rest it is extension. The position of rest of the foot, translated into the language of orthopædics, is *talipes calcaneo-valgus*. In adduction of the foot the plantar arch was increased—*pes cavus*; in abduction it is flattened—*pes planus*. The toes are straight at their metatarso-phalangeal joints, or even a little flexed towards the sole. The interphalangeal joints are also straight; perhaps the distal one may be extended. Hence here is the opposite condition to the digiti extensi of the position of activity—digiti flexi. The big toe is also straight, and when stiff and painful is called *hallux rigidus*. Should the condition increase, the big toe becomes flexed at the metatarso-phalangeal joint—*hallux flexus*. The position of the big toe is one of abduction with the rest of the foot—*hallux valgus*. But as this position of the toe is produced by "mesially pointed boots," it is found in other conditions than the resting position of the foot.

From this it is possible to derive certain rules, such as was the case for the position of activity.

(1) Inactive and relatively weak people usually turn their toes out.

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(2) Those who habitually turn their toes out have some degree of pes planus and small calf muscles.

(3) The boot keeps its shape, the upper not being creased transversely.

(4) The boot is worn unevenly, particularly on the outer side of the heel.

(5) The skin on the sole of the foot is harder, particularly so on the outer side of the heel and under the third toe. It is often soft and silky under the big toe.

(6) The position of rest is associated with tendencies to the following deformities: (1) talipes calcaneus, (2) talipes valgus, (3) pes planus (4) hallux valgus, (5) hallux rigidus, (6) hallux flexus, (7) digiti rigidi, (8) digiti flexi.

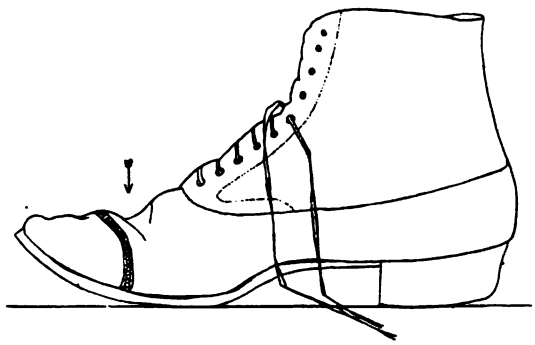

FIG. 2A.—The boot of a patient with pes cavus, showing the curling up of the toe and the transverse creasing.

THE RELATIONSHIP BETWEEN MOVEMENTS AT THE ANKLE, MID-TARSAL, AND OTHER JOINTS OF THE FOOT.

There is a very distinct and intimate relationship between the movements at these joints. Movements are possible at one joint quite independently up to a certain extent. But beyond this point combined movements at more than one joint can alone take place. For instance, a foot is capable of some inversion or eversion at the mid-tarsal joint without any movement taking place at the ankle-joint. But such rotatory movements are very limited. Marked inversion of the foot can only be accomplished if the ankle is flexed, and marked eversion is only possible with extension of the ankle-joint. In the first place the foot takes the position of *talipes equino-varus* and in the second that of *talipes calcaneo-valgus*. Extensive rotatory movements of the foot can only be accomplished if combined with some movement at the ankle-joint.

But rotatory movements are intimately associated with the condition of the arch of the foot—inversion and adduction with an arched instep—eversion and abduction with a flattened instep. Further, the condition of the arch of the foot exerts considerable influence upon the movements at the metatarso-phalangeal joints of the toes. In fact, movements of one joint of the foot affect all the other joints. So that a deformity which appears to affect but one joint in reality affects all parts and joints of the foot. There is no such thing as a simple and single deformity of the foot. This is a very important point in recommending treatment for a particular case; because one has the choice

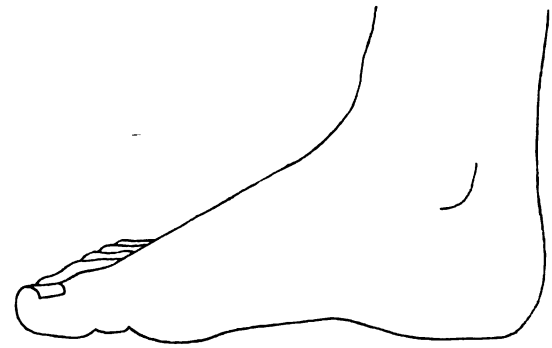

FIG. 3.—The foot in rest.

of affecting, by instrument or operation, the deformity, directly or indirectly, according to which portion of the foot is attacked. Further, one must always consider that an operation upon one part or joint will always affect the rest of the foot. In this way undesirable results may follow an apparently obviously indicated operation. Or by means of some indirect and not obviously indicated procedure it may be possible to give the patient a useful foot.

PES CAVUS OR PES ADDUCTUS.

An arched instep is an integral part of the position which the foot assumes in activity. Pes cavus, like every other deformity, may be congenital or acquired. The congenital form is practically only seen as a part of congenital talipes equino-varus, but it may be recognised in another and more or less congenital form. The deformity of talipes equino-varus consists of two parts, the equinus and the varus. The equinus takes place at the ankle-

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joint and, owing to the tendo-achillis, is difficult to reduce; the varus takes place mainly at the mid-tarsal joint and is comparatively easily reduced by manipulation. In consequence, if the degree of the deformity is not great, when the child is taught to walk it corrects the varus, leaving behind some small degree of equinus. It has been shown that cavus is an integral part of varus. Correction of the varus by manipulations rather increases than diminishes this cavus. It seems probable that very many, if not all, cases of high instep are really cases of completely or incompletely corrected talipes varus or equinovarus.

Pes cavus is, then, a deformity the seeds of which are sown in intra-uterine life, and which is perpetuated by the growth of the bones adapting themselves to the position. This is the probable origin of all cases of pes cavus which are associated with any degree of equinus, such as right-angled talipes equinus, and also possibly of others in which the degree of varus preponderates over that of the equinus. This explanation puts the pathology of both congenital and acquired pes cavus on quite a different footing to that which it has previously occupied. Parkin, Clutton, and others attributed the cavus to the equinus, but observation teaches us that it is dependent on the varus which is so very commonly associated with the equinus. It is exactly the same with acquired cases of pes cavus, which is a complication of every case of acquired talipes equinovarus, being produced in most cases by infantile paralysis, anterior poliomyelitis.

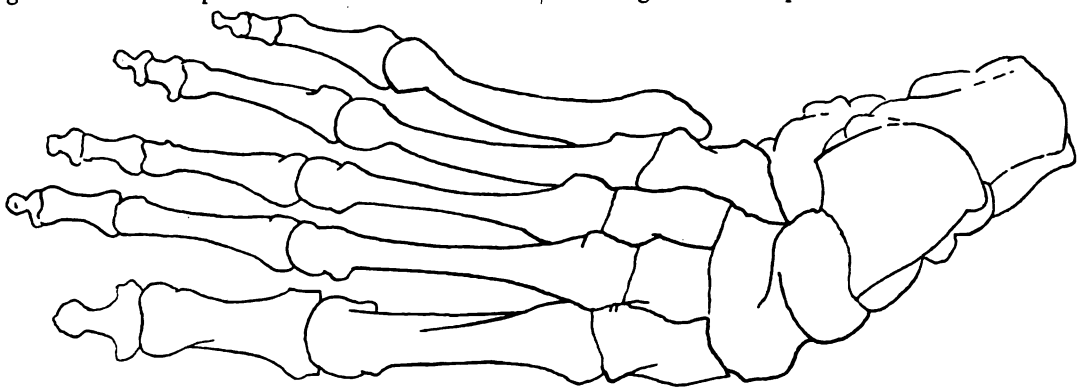

Fig. 4.—The bones of the abducted foot.

Pes Planus or Pes Abductus.

Just as pes cavus was derived from the position of activity assumed by the foot, so is its opposite, pes planus, derived from the opposite position of the foot, namely that of rest. The position of activity is by far the most frequently assumed by the foot at birth; that of rest is uncommon. In consequence, pes planus is an uncommon condition to find congenitally. But, in truth, it is a deformity which is acquired early in life. If the general health and habits of the child make it weakly and cause it to lead an inactive life, the foot assumes the position of rest, abduction or talipes calcaneo-valgus, as that posture requires no muscular effort to support it. Both the hard and soft growing structures of the foot soon render this the habitual position; and as the bodily conditions and occupations of the patients render them incapable of correcting the position, it soon becomes a permanent deformity, getting worse as further growth takes place.

Examples of Abducted Feet with Symptoms of Flat Foot but which have an Arched Instep.

It will be seen that the origins of both pes planus and pes cavus are to be found in early life, the latter not infrequently in intra-uterine life. Amongst over a thousand patients, the late Mr. Walsham found that over three quarters of the cases of pes planus were met with under twenty years of age, the deformity being by far the most frequent at the ages of fourteen and fifteen. Clinical observations on cases of pes cavus lead one to a similar conclusion, namely, that both of these deformities commence in childhood, becoming sufficiently advanced and noticeable as to be brought to the surgeon's notice in early adult life. But I would point out a further and most important clinical fact which has

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hitherto escaped notice. In practice one sees a number of patients who complain of all the symptoms of flat foot, yet when examined they have a somewhat arched instep and not the flat foot as it is described. Sometimes these insteps present quite considerable arches. These cases are at first sight difficult to understand. But if one reflects for a moment the explanation will be obvious. An exaggeration of the position of the foot in activity leads to talipes equino-varus and

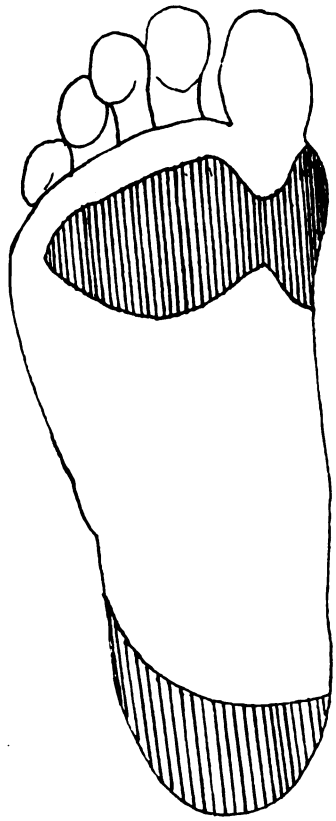

FIG. 5.—Diagram of the corns on the sole of the arched foot.

pes cavus; and an exaggeration of the position of rest to talipes calcaneo-valgus and pes planus. The foot is normally in a position of activity. Let us consider the case of a patient who naturally has a high instep—*i. e.* a condition of some pes cavus associated with little or no talipes equino-varus. The foot is in an exaggerated position of activity, though this is not deemed always pathological. Every foot will have its own particular positions for activity, adduction, and for rest, abduction. But as the foot has a high instep—*i. e.* is by nature adducted already more than is usual—it will be unable to undo all its natural adduction when it assumes the position of rest. Therefore, though abducted and at rest, there will be an arch in the instep. A more ordinary foot with a less high instep and, therefore, less adducted, will be able to undo its arch and its natural adduction completely. It will now be seen that every foot, in a degree corresponding to that of its natural arch, has a position of abduction

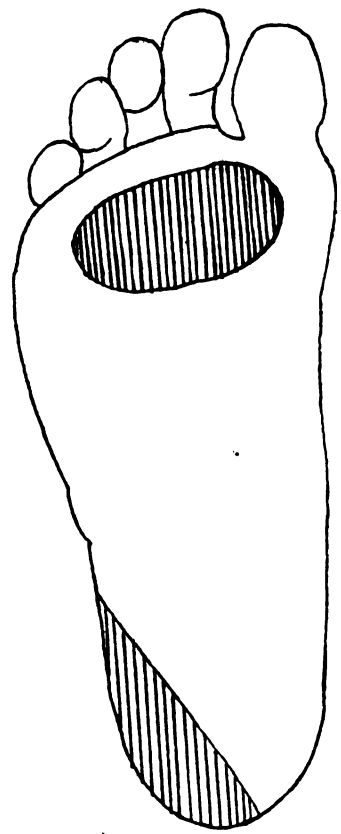

FIG. 6.—Diagram of the corns on the sole of the flattened foot.

or a degree of "flat" foot of its own when resting. In some cases the natural arching of the instep is not obliterated when the foot is in the position of rest. So that there are examples of the so-called pes planus which having some arch are not flat, although patients may complain of all the symptoms and exhibit most of the signs of so-called flat foot.

A second example can be quoted to show that every foot, according to its degree of arch or activity, has a corresponding degree of flatness in

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rest. The high-heeled boot, so popular amongst ladies, places the foot in a position with raised heel and arched instep—*i. e.* talipes equinus (Fig. 7). The mesial point of the boot displaces the big toe outward into a position of hallux valgus. As I said before, hallux valgus is naturally associated with the position of rest with flattened arch, talipes calcaneo-valgus and pes planus, not with that of activity with arched foot, talipes equino-varus and pes cavus. Yet, although ladies' feet are distinctly arched, they have halux valgus, an apparent contradiction. An explanation, besides that of the boots, is to be found by studying the callosities on the sole of the foot; in pes cavus there is a callosity on the inner side under the ball of the great toe which is not present in pes planus (see Figs. 5 and 6). If the soles of ladies' feet are examined, it will be found that the skin under the ball of the great toe is frequently "thin and silky"

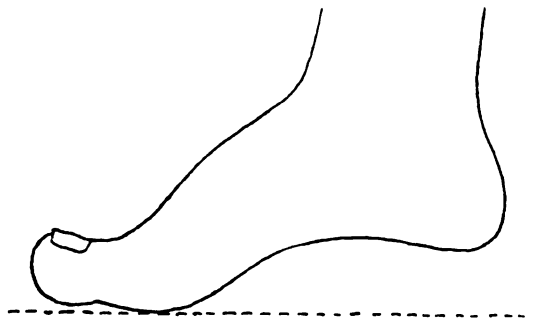

FIG. 7.—A lady's foot.

whilst under the third toe it is hard and horny. So that, in spite of its equinus and apparent arch, the foot is abducted into a position of rest and should be called "flat" or its equivalent if it gives rise to symptoms.

The study of these two types of foot has made it abundantly plain that the name pes planus is not a good one. It is used to denote the condition of flat foot and its symptoms. But there are patients who present all the symptoms of "flat foot" but whose feet possess an arch; and though they are not anatomically flat, they are so physiologically. Pes planus is a bad name, meaning two different things when considered physiologically or anatomically. The trouble which the patient complains of is not concerned with the arch of the foot, cavus or planus, but with the fixation of that foot in the adducted or abducted position, commonly called varus and valgus position. I suggest that the names "pes cavus" and "pes planus" be dropped and that "pes adductus" and "pes abductus" be retained for the conditions, as they explain the pathology and natural history of the deformities.

(*To be continued.*)

August 27th, 1906.

MECHANISM OF THE THERAPEUTIC ACTION OF NITRITE OF AMYL.

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IT has been amply demonstrated that nitrite of amyl causes widespread, if not general, peripheral vaso-dilation, and it is the object of this article to show that this action is alone sufficient to explain the therapeutic effects which have been, or may be, observed in a number of clinically diverse affections.

The general or aortic blood-pressure depends upon the energy of the heart and upon the peripheral resistance in the vascular system. The maintenance of this pressure at an approximately uniform mean height is admitted to be one of the main objects of the circulatory mechanism. And, further, it is admitted that this object is attained during physiological life. But the peripheral resistance in the vascular system depends for the most part upon the degree of constriction or tone of the smaller arteries. And it is known that this tone is ever varying in different parts of the organism in accordance with the ever varying rise and fall of functional activity therein. It follows "that every variation in one part is compensated by a simultaneous and contrary variation in another part" (Leonard Hill).* Were this not so, it is obvious that the object of the arterial dilation—namely the increased supply of arterial blood that occurs in every organ or tissue which is actively functionating—would be in part defeated. For an uncompensated vaso-dilation would reduce the general blood-pressure and so tend to diminish the local blood-supply. It follows, therefore, that the compensatory vaso-constriction is always an im-

* 'Text-book of Physiology,' Schäfer, vol. ii, pp. 81, 82.

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portant reinforcement in securing the increased blood-flow through the dilated artery; in other words, any *local* increase in blood supply depends in great part upon the due maintenance of *general* arterial tone.

But variations in vascular tone may be too widespread and intense to permit of adequate compensation by the contrary vascular variation elsewhere. Then so dominant is the necessity for uniformity in general or aortic blood-pressure that variations in cardiac action are superadded. A vaso-constriction too widespread and intense to permit of adequate compensation by vaso-dilation elsewhere, is accompanied by cardiac inhibition through the vagus: a vaso-dilation too widespread and intense to permit of adequate compensation by vaso-constriction elsewhere is accompanied by cardiac augmentation through the augmentor nerves. As Leonard Hill says: "If all the cardiac nerves be intact, a rise of arterial pressure always slows the heart, and a fall accelerates it."* This mechanism was first recognised by Marey.

It is my opinion that the above physiological principles may be applied with distinct advantage to the investigation of numerous pathological problems. If slowing of cardiac action is observed, we should in the first place search for some widespread area of vaso-constriction; if acceleration, then for some widespread area of vaso-dilation. If without variation in cardiac action vaso-constriction is observed, we should search for compensating vaso-dilation; if, in the same circumstances, vaso-dilation is observed, we should search for compensatory vaso-constriction. *A fortiori*, vaso-constriction associated with cardiac acceleration and vaso-dilation associated with cardiac slowing, should impel us to search for the contrary vascular conditions. It may be that we shall often fail; in that case no harm will have been done. But it is certain that we shall not infrequently succeed, and that such success will throw an unaccustomed light on many obscure processes, physiological and pathological.

Menstruation.—During menstruation there is admittedly marked vaso-dilation affecting the generative organs, external and internal, and, to some extent, the pelvic viscera generally. Concurrently—though this is not often insisted on—there is a compensatory exaggeration of the normal peripheral vaso-constriction or physiological tone in other areas. This might be deduced, but has been demonstrated by Oliver (amongst others), who compares the constricted condition of the radial artery during menstruation to that which is often found in the gouty and during migraine.* The joint effect of these complementary vascular changes will be to secure an intense vascular distension—in older but quite accurate phraseology, a "determination of blood"—in the genital organs. And since this vascular distension falls in great part upon the uterine mucosa, which at this stage is newly formed, friable, and probably about to undergo disintegration, the natural result is the menstrual flow, which relieves the distension.

From this view it is easy to deduce the result of an inhalation of nitrite of amyl given during the menstrual flow. Such would, indeed, tend to reverse the action of the menstrual mechanism. There would be, consecutively, widespread peripheral vaso-dilation, decrease of vascular distension of the uterine mucosa, and decrease or cessation of the menstrual flow. I had long reached this deduction but hesitated at verification, holding as I do strong views as to the danger of suddenly checking the menstrual flow. Eventually, however, verification came accidentally. Being called to a lady in an anginal seizure, I administered a capsule of nitrite of amyl. The result was immediate relief from the pain. But some days later I learned that the menstrual flow which had been present at the time had ceased simultaneously with the relief from the pain, and had not returned. The patient then informed me that her anginal seizures occurred almost invariably at her menstrual periods, that nitrite of amyl had been prescribed by many medical men, and that the drug had practically always terminated the period along with the paroxysm. She added that she usually suffered thereafter from a series of paroxysms. These she ascribed, with justice in my opinion, to the stoppage of the flow by the drug.

Menorrhagia.—The above case was reported in the 'Lancet,'† whereupon Dr. Horace C. Colman argued that a drug which stopped normal menstruation would probably check the excessive flow in menorrhagia. His anticipation was tested and

* *Ibid.*, p. 56.

* 'Brit. Med. Journ.,' 1896, vol. i, p. 1375.

† 'Lancet,' 1904, vol. ii, p. 522.

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verified in a case reported in the 'Scottish Medical and Surgical Journal.'* In three consecutive menstrual periods nitrite of amyl was administered. In each instance the flow was at once greatly diminished, and ceased within twelve hours. No ill effect followed, and there ensued a material improvement in the general health.

Dysmenorrhœa.—Nitrite of amyl is known to afford relief in some cases of dysmenorrhœa,† and very often such relief is explained by the relaxation of uterine spasm. But I can find no demonstration that the nitrites in medicinal doses have any relaxing influence upon muscular fibre other than that which constitutes the main portion of the middle arterial tunic. Further, in some of my cases the pain was distinctly ovarian in character, and in all in which relief followed there was a diminution, sometimes a cessation, of the flow. Hence it is reasonable to believe that the relief depended on the diminution of pelvic vascular distension—which must, of course, be a factor in dysmenorrhœa, as in eumenorrhœa—and that the diminution of pelvic vascular distension depended on the reduction of peripheral vaso-constriction.

Hæmoptysis and other deep-seated hæmorrhages.—From the rapid hæmostatic influence of nitrite of amyl on the normal and exaggerated menstrual hæmorrhage, we may infer a similar action on many other hæmorrhages from the systemic arterial system. It would be surprising, for example, to learn that the administration of the drug was without some immediate salutary influence in hæmatemesis, whether dependent on, or independent of, gastric ulceration, in hæmorrhage from internal malignant disease, and in other inaccessible hæmorrhages, non-traumatic and traumatic. And Dr. C. F. A. Moss has recently published two cases of severe internal hæmorrhage in the first of which nitrite of amyl "acted like a charm." One was a case of reactionary hæmorrhage following the removal of bilateral parovarian cysts. The other was a case of hæmorrhage from rupture of the sac of an extra-uterine gestation.‡

But the hæmostatic utility of the drug will not be limited to hæmorrhages from the systemic arteries. The fall of blood-pressure from vaso-dilation of the systemic periphery will travel backwards in the course of the circulation into the left ventricle, left auricle, and pulmonary periphery; wherefore cases of hæmoptysis, phthisical, mitral, or other, will be susceptible of relief by the drug. In the pages of the 'Lancet'* I have recorded nine cases from various sources in which inhalation of nitrite of amyl (on all occasions but one in which relief was but partial) immediately stopped the bleeding. Since then Dr. Horace C. Coleman reports an equally satisfactory case.† Dr. C. Reissman, medical officer in charge of the Kalyra Sanatorium, and of the department for advanced cases of phthisis in the Adelaide Hospital, South Australia, writes: "What you say about the use of amyl nitrite I have found to be perfectly true from my own observation, and following your directions, I order every patient who has had or may have hæmoptysis, to carry a bulb of amyl nitrite with him.' And the 'Lancet's' Paris correspondent states‡ that M. Rouget reported a series of ten cases in which tuberculous hæmoptysis was treated in this way; the results were remarkable; in one case the hæmorrhage could not be checked by any other remedy.

Rigor.—Exposure of the body surface to cold is followed by a series of complex vascular changes, all of which are conservative and adapted to prevent a fall of body temperature. There is cutaneous anæmia from vaso-constriction. Thus there is a saving of heat loss. When the cold is intense there is increased tonus of the muscles of the body;§ and if the exposure is prolonged the increased tonus develops into actual rigor. Thus there is an increase of heat-production. Now, since the muscular layer of the body in rigor is in extreme functional activity, there can be little doubt that the muscular arteries are dilated. And such vaso-dilation may be regarded as compensatory of the cutaneous vaso-constriction. For the relief of such rigors a hot alcoholic drink is commonly prescribed with success. But it will be found that an inhalation of nitrite of amyl, if less pleasant, is even more rapidly successful. With both drugs the vascular mechanism of the rigor is reversed; there is cuta-

* 'Scottish Medical and Surgical Journal,' May, 1905.

† 'Practical Therapeutics,' H. A. Hare, 1900, p. 72.

‡ 'Lancet,' October 14th, 1905, p. 1107.

* *Ibid.*, 1904, vol. ii, pp. 522 *et seq.*, 942 *et seq.*, 1446; 1905, vol. i, p. 187.

† 'Scottish Medical and Surgical Journal,' May, 1905.

‡ 'Lancet,' May 6th, 1905, p. 1236.

§ Winternitz, in von Ziemssen's 'Handbook of Therapeutics'—"Hydrotherapeutics," p. 429.

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neous vaso-dilation, with concurrent relief of muscular vascular distension.

But rigors occur in various specific diseases. Here, though causation differs, the vascular mechanism is identical. In the malarial rigor there is intense cutaneous vaso-constriction; in severe cases the radial may be reduced apparently to the size of a piano wire with imperceptible pulsation. At the same time cardiac action is accelerated. Hence there must be some extensive area occupied by vaso-dilation. Obviously this area is the functionally active muscular layer, the arterioles of which are collectively "so large that the blood is able to escape into the veins as rapidly as through the vessels of the splanchnic and skin areas together" (Lauder Brunton).* It may be objected that the vascular distension of the muscular area in rigor is a mere unverified inference. But there are cases recorded which practically amount to a demonstration. For example:

A soldier had spent several years in India, where he had suffered severely from intermittent fever. He developed disease of the left knee-joint and was invalided home in consequence. Being very weak and emaciated, amputation through the thigh was performed on July 8th. On July 16th the temperature and pulse rose, and the stump became swollen. This change was accompanied by cold and hot stages resembling ague: on the 17th there was oozing of blood from the stump, which was opened up for the application of styptics. The hæmorrhage, however, recurred, on the 18th, 20th, 21st, and 23rd. On each occasion it was preceeded by cold and hot stages, and on each occasion it was found necessary to open the stump and apply styptics. The patient became extremely pale and almost pulseless. Five grains of quinine were then given thrice daily. The bleeding did not recur and the patient made an excellent recovery. (Reported by Surgeon-Major Porter).†

There can be little doubt that this patient suffered from a recrudescence of malaria such as is prone to occur after any operation or traumatism in those who have previously suffered, and that the intermittent hæmorrhage was due to intermittent vascular distension of the cut muscles. The radical treatment in such cases must, of course, be by quinine; but inhalation of nitrite of amyl would almost certainly have checked the hæmorrhage for the time being.

That malarial and other specific rigors can be immediately modified or stopped by the nitrite is easy to show. I have recorded cases in point under the care of Drs. C. S. Hawkes and W. J. Fearnley;* and Dr. W. H. Rand refers to many in the pages of 'American Medicine.'†

Probably in most cases the only advantage gained would be the relief of discomfort. But in the algide stage of pernicious malarial fever the pulse is infrequent instead of being frequent, and sometimes the arterial spasm "may be carried to such a degree as to bring the heart to a standstill," and so lead to fatal syncope (Broadbent).‡ Here we may infer that the muscular vaso-dilation, if present, is inadequate for compensation, and that cardiac inhibition through the vagus is substituted or superadded. Nor is it difficult to imagine that nitrite inhalation might then prove a life-saving measure.

Angina pèctoris.—Many views are entertained as to the exact local mechanism of the pain in angina. The pain has been ascribed to intermittent claudication of the coronary arteries (Huchard), to cramp or strain of the heart, and to other conditions. For myself I have argued that it depends on vascular distension in the mediastinum due to vaso-dilation probably of the coronaries and adjoining arteries supplying the cardiac plexus, which vaso-dilation is compensatory of vaso-constriction elsewhere.§ But whatever view be taken, it must be admitted that in most cases, if not in all, an essential factor is an increase of peripheral resistance from exaggerated vaso-constriction. And this is the only factor which need be considered in this connection. As Lauder Brunton long since demonstrated, nitrite of amyl terminates the anginal paroxysm by promoting sudden peripheral vaso-dilation.‖

Syncope.—Brain anæmia directly dependent on

* 'Lancet,' April 11th, 1903, p. 1056.
† *Ibid.*, February 26th, 1875, p. 313.

* 'The Food Factor in Disease,' Francis Hare, vol. i, p. 295. (Longmans, Green & Co.).
† 'American Medicine,' April 29th, 1905, p. 682.
‡ 'The Pulse,' 1890, pp. 189, 153.
§ 'The Food Factor in Disease,' 1905, vol. i, p. 316 *et seq.*
‖ 'Pharmacology, Therapeutics, and Materia Medica,' 1885, p. 710 *et seq.*

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weak cardiac action is admittedly the proximate cause of syncope (excluding syncope from hæmorrhage). But it is permissible for us to go farther back and search for the proximate cause of the cardiac weakness. Now, Goodhart has expressed his belief that many swoons are "largely a matter of peripheral spasm";* and a sudden widespread vaso-constriction might well demand compensatory cardiac inhibition through the vagus. In this way we could readily explain the syncope which may accompany fright or mental shock, and that which may occur during cold bathing; in both cases the peripheral vaso-constriction would be cutaneous. In this way we could explain the syncope which may accompany violent paroxysmal cough;† in this case the vaso-constriction would probably be splanchnic as in asphyxial conditions generally. And in no other way, so far as I can see, is it possible to explain the instantaneous effect of nitrite of amyl in many syncopes. I have recorded several cases in which a state of syncope dependent on pain, mental impressions, etc., was by this drug immediately relieved synchronously with the flushing of the surface. ‡

Hay fever.—Whatever the dominant factor in this common affection—whether the disease depends essentially on pollen or other inhaled stimulants, on malassimilation of food,§ on eye-strain, ‖ or what not—there can be no doubt that the prominent symptoms are the direct result of vascular distension of the nasal mucosa. In this, as in all vascular distension, there are at least two essential factors, namely, local vaso-dilation and general vaso-constriction, which latter may not, of course, amount to more than physiological vascular tone. Hence there are at least two means of affording relief—namely, the promotion of local vaso-constriction, and the promotion of general vaso-dilation. The following case illustrates the efficiency of both means:

The wife of a medical man, conversant with these views, suffers greatly from hay fever. She frequently uses adrenalin spray, and thereby obtains immediate, though temporary, relief. But one night when suffering from nasal block to such an extent as to preclude sleep, she found herself without any adrenalin in the house. At her husband's suggestion she inhaled a nitrite of amyl capsule. Synchronously with the appearance of the cutaneous flush the nasal passage became quite free and remained so sufficiently long to enable her to get to sleep.

Asthma.—In a recent work ('The Food Factor in Disease,' Longmans, Green & Co.) and elsewhere,* I have argued that the obstructive dyspnœa of asthma depends, not upon constriction of the smaller bronchi through contraction of their circular muscular fibres, but upon vascular distension of the lining mucosa of these air-tubes. This vascular distension, though differing widely in location, is identical in nature with that which occurs in hay fever. Moreover, it depends proximately upon the same mechanism—namely localised vaso-dilation combined with widespread vaso-constriction in other areas. But since the localised vaso-dilation of asthma, unlike that of hay fever, is extensive, it follows that the widespread vaso-constriction demanded for compensation must be marked and will, therefore, be perceptible. Hyde Salter demonstrated that "the pulse during severe asthma is always small, and small in proportion to the intensity of the dyspnœa."† He never knew the small pulse absent in severe asthma, and he pointed out that "immediately the paroxysm yields the pulse resumes its normal volume."‡

Some of those who took part in a recent discussion on asthma expressed surprise that both vaso-constrictors such as adrenalin and vaso-dilators such as the nitrites should have been confidently recommended in the treatment of the asthmatic paroxysm. But in asthma, as in hay-fever, both classes are capable of affording relief since both are capable of reducing vascular distension though operating in different ways. Inhalation of adrenalin spray has been shown to give relief in asthma. Here vascular distension is reduced through localised vaso-constriction. Adrenalin by the mouth or hypodermically also may be effectual. Here vascular distension is reduced through *general* vaso-constriction, and the onus of compensation is thrown upon the heart, the action of which is

* 'Lancet,' 1901, December 21st, p. 1715.
† *Ibid.*, p. 1714.
‡ 'The Food Factor in Disease,' 1905, vol. ii, p. 132.
§ 'Brit. Med. Journ.,' 1905, July 15th, p. 161.
‖ *Ibid.*, August 26th, p. 435.

* "The Mechanism of the Paroxysmal Neuroses," 'Australasian Medical Gazette,' August, 1903.
† 'On Asthma,' 1868, p. 72.
‡ *Ibid.*, p. 73.

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slowed. Finally, inhalation of nitrite of amyl is "invaluable in many cases" (H. A. Hare),* and rarely fails to give relief from dyspnœa. Here vascular distension is reduced through the promotion of vaso-dilation in the widespread areas of vaso-constriction. Naturally those who approach this subject imbued with the pre-conviction that asthma depends upon bronchial constriction will conclude that the nitrite operates by relaxing this constriction, but, as already mentioned, there is no independent evidence that the drug possesses any such capacity.

Some neuralgic affections.—Much evidence can be adduced to show that many neuralgic conditions depend proximately upon vascular distension of nerve-trunks, ganglia, or the tissues in which the nerves are imbedded.† As in the cases already considered, such vascular distension depends upon two factors—namely vaso-dilation in the arteries leading to the seat of pain and vaso-constriction elsewhere, which latter may, or may not, be exaggerated beyond the normal. On this view only, is it easy to explain the sudden relief from pain which so frequently follows the inhalation of nitrite of amyl. Lauder Brunton says: "As migraine is generally connected with vascular spasm, I employed the nitrite of amyl in headache and found that frequently, though not invariably, it relieved the pain."‡ Dr. C. S. Hawkes, of Brisbane, gave the nitrite in a case of gastralgia in which pain had been nearly constant for a week: instant relief lasting two hours ensued. I have myself obtained similar results in hemifacial and brachio-cephalic neuralgia, in neuralgia of the pubes and urethra, and in one case of sciatica.

Epilepsy.—Though long under a dense cloud, the vasomotor theory of epilepsy—the theory that the fits in some cases depends upon sudden brain anæmia due to spasm of cerebral arteries—has never been fully obscured: it has been held throughout by many clinicians. For some time, it is true, the cerebral arteries were thought to be without vasomotor nerve-supply. But that error has been corrected; it has now been shown by Gulland and others that the cerebral vessels are surrounded by the usual perivascular nerve-plexuses.*

But in the work already referred to I have pointed out that the probability that in some cases the sudden brain anæmia responsible for the fit depends upon a more complex circulatory disturbance than a mere vaso-constriction more or less localised in the cerebral area. I have argued that in these cases there is a widespread vaso-constriction throughout the organism, which, from the suddenness of its onset and from its extent, precludes adequate compensation by vaso-dilation, and, consequently, demands cardiac inhibition through the vagus; and that the partial or complete interruption in the circulation so brought about—which, indeed, has been frequently observed†—is in these cases the proximate factor in the sudden essential anæmia of the brain.

Now, on both of these theories—but not any theory which excludes a pathological degree of vaso-constriction as an essential factor in the epileptic fit—can the undoubted influence of nitrite of amyl in epilepsy be explained. Crichton Browne and others have shown that when administered immediately after the appearance of the aura, nitrite of amyl prevents the fit which would otherwise have come on.‡ The commencement of the aura presumably marks the commencement of the disordered vasomotor action; and the administration of the nitrite paralyses this action, thus rendering impossible the development of the subsequent circulatory changes, whether these are restricted to the vessels or include modification of cardiac action.

Conclusion.—In all the affections referred to in this article, whether their prominent clinical manifestations depend upon vascular distension or upon anæmia, directly or indirectly from vaso-constriction, it is, I think, permissible to conclude provisionally that the rapidly favourable action of nitrite of amyl depends upon the promotion of peripheral vaso-dilation. And if we are justified in this conclusion, then it follows that the drug will be of material use in many other affections of similar mechanism—for example, in the emergency of impending asphyxia in some cases of non-diphtheritic croup, and in that which is liable to occur in angio-neurotic œdema affecting the oral, faucial, pharyngeal, and laryngeal mucosæ.

August 27th, 1906.

* 'Practical Therapeutics,' 8th edition, p. 492.

† 'The Food Factor in Disease,' 1905, vol. i, p. 382, *et seq.*

‡ 'Pharmacology, Therapeutics, and Materia Medica,' 1885, p. 711.

* 'Text-book of Physiology,' Schäfer, 1900, vol. ii, p. 168.

† Moxon's "Croonian Lectures," 'Lancet,' 1881, vol. i, p. 649 *et seq.* See also an article by A. E. Russell entitled "Cessation of the Pulse during the Onset of Epileptic Fits," 'Lancet,' July 21st, 1906, p. 152.

‡ 'Pharmacology, Therapeutics, and Materia Medica,' Lauder Brunton, 1885, pp. 711, 712.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A LECTURE

ON

DILATATION OF THE STOMACH.*

By ROBERT HUTCHISON, M.D., F.R.C.P.,

Assistant Physician to the London Hospital, and to the Hospital for Sick Children, Great Ormond Street.

LADIES AND GENTLEMEN,—By dilatation of the stomach I mean enlargement of the organ, along with delay in the transmission of its contents onwards. The term "dilatation," then, you will see rightly involves two conceptions, one which is anatomical, enlargement, and the other physiological, delay in the transmission of contents onwards. Enlargement of the stomach alone does not necessarily mean dilatation, because one meets occasionally with large stomachs in gross feeders, stomachs which are quite able to drive their contents onwards in the normal time and without any discomfort to the patient. Nor, on the other hand, has one any right to call a stomach dilated in which the contents are delayed in their transmission unless also the organ be enlarged.

Two Forms of Dilatation.

Now, enlargement of the stomach, with delay in the transmission of its contents—in other words, dilatation—may be due to one of two conditions. It may be primary or it may be secondary. When primary it is due to conditions inherent in the stomach-wall itself; when secondary it is due to some obstruction to the outlet. It will be convenient if we use different terms for these two varieties of dilatation, and speak of the first as atonic dilatation and of the second as obstructive dilatation. Now, atonic dilatation rarely, if ever, produces a really large stomach. I think that the characteristic features of this form are a stomach

* Delivered at the Medical Graduates' College and Polyclinic.

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which is somewhat enlarged and which passes on its contents slowly, but yet in which there is no actual stagnation of contents as manifested by fermentation, and for that reason there are some who hold that one should not speak of primary dilatation as dilatation at all, but that it is better to speak of it as distension, or, as some even prefer to call it, "extension" of the stomach, because it is so different in its characters from the obstructive or secondary form. At all events, whatever name you apply to it, I wish to say that the atonic form rarely, if ever, results in the very much enlarged stomach, with fermentation of contents. Secondary dilatation gives rise to the most typical form of dilated stomach—that is, a greatly enlarged organ with retention and stagnation of contents, accompanied by fermentation and all the other signs which one associates with classic examples of dilatation. Secondary or obstructive dilatation may be due to several causes. Amongst these are congenital pyloric stenosis, benign or malignant stricture, pyloric spasm, and pressure or traction upon the pylorus from without.

As regards the causes of primary or atonic dilatation of the stomach I would only say that it is apparently due, in many cases at least, to an inherent or congenital weakness in the stomach itself—that is to say, in the stomach wall. There are people—one sees it often in young subjects—who seem to have been born with a congenitally weak stomach, one which is easily over-distended, and just as one meets with people whose hearts are readily dilated on any abnormal exertion, so I think there are some people whose stomachs are abnormally extensible. One finds very good examples of it after some acute exhausting illnesses. I have seen several examples of it, for instance, after typhoid fever, and indeed the frequency with which it occurs after typhoid fever has been used as an argument in favour of careful feeding during convalescence from that disease, the danger of over-stretching the stomach being so great. Far more commonly, I think, atonic dilatation is associated with an acquired condition of neurasthenia, and is really only a part of the general "want of tone," if you like to use that term, which is known to exist throughout the body as a whole in neurasthenia. You will find that these patients are often people who have been overworked, or subjected to some great strain, mental or bodily, or both, and who exhibit various signs of nervous exhaustion.

Physical Signs of Dilatation.

I now wish to speak for a moment of the methods by which one recognises the existence of dilatation. It may be said that the diagnosis of dilated stomach can but rarely be made from symptoms alone, for I suppose that it would be agreed that the only pathognomonic symptom of dilatation of the stomach is the vomiting of food which can be identified as having been swallowed a long time before. Apart from that, which one rarely has an opportunity of seeing, there is no pathognomonic symptom of dilatation, and hence one has to depend upon the results of physical examination for its recognition.

Now, the greater curvature of the stomach should lie about the level of a line drawn between the tips of the tenth ribs on each side. This is more accurate than giving its relation to the umbilicus, for the umbilicus is far from being a fixed point, and in the majority of cases the above line lies considerably above the umbilicus, usually at least two fingers' breadths above it. Hence if the greater curvature should extend below the umbilicus the stomach may be looked upon as definitely dilated. Now, the difficulty often is to determine where exactly the greater curvature lies. One can sometimes, if one is fortunate, make it out by means of simple percussion from below upwards. But the difficulty associated with that method is the frequency with which the colon is distended with gas, so that it is not always easy to distinguish between the stomach note and the colonic resonance. Various means have been introduced for overcoming this difficulty. One can sometimes get over it by inflating the stomach, and, in spite of certain disadvantages which it entails, this is an exceedingly trustworthy method of making out the exact limits of the stomach resonance. Inflation is best carried out by administering effervescing powders, which obviates the necessity of passing a stomach-tube. Careful observations have been made as to the exact degree to which the stomach allows itself to be distended. If, for instance, you administer 75 gr. of tartaric acid and follow that up by 120 gr. of bicarbonate of soda, it has been found that the distance between the lesser and greater curvature of the normal stomach increases

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to the extent of 2 cm. A stomach which is displaced—that is to say, in which there is gastroptosis—enlarges somewhat more, about 3 cm., and an atonic stomach enlarges about 5 cm. If the stomach enlarges beyond that it is not merely atonic but it exhibits that degree of dilatation which is usually associated with obstruction.

Another method which is sometimes used with great advantage is to make the patient swallow a glass or two of water, and then to percuss out the dulness so produced from below upwards. I do not know that that is not as good a method as any for making out the exact position of the greater curvature. It is especially useful where there is a danger of confusing the stomach resonance with that of colon. Of course in that case percussion must be carried out with the patient standing or sitting up. The water collects in the greater curvature, and you percuss out, without any difficulty, the line of dulness that it forms. Instead of ordinary water I often use effervescing water, because the gas which it contains distends the fundus of the stomach, and one is thus able to percuss out the fundus distended with gas and the greater curvature full of fluid, so that not only the lower but the upper limits of the stomach can be made out. The delimitation of the upper limit or fundus is especially useful in the atonic form of dilatation, because in that form the dilatation is often mainly upwards, and it may extend as high as the fourth space, which, of course, is much above what it ought to be, the space between the fifth and sixth ribs being the normal upper limit of the fundus. Or it may pass backwards beyond the mid-axillary line, which is the usual posterior limit of fundus resonance. The condition which one has especially to diagnose from dilatation is a displacement downwards of the stomach. You know that it happens not uncommonly that the stomach assumes a horizontal position (what is called the "sling stomach"), or it may assume a more or less vertical position in the left of the abdomen, the whole pyloric portion having descended. You will find in such circumstances that there is no note over Traube's area as there should be, and you can percuss out the organ as a whole, showing that the lesser curve has descended from the normal position, and is not further removed than it should be from the greater curvature. The distance between the lesser and greater curvatures is, normally, 4 cm., and anything beyond that means dilatation and not mere displacement.

I must now speak of a "splash" as a sign of dilatation of the stomach. Inferences from the presence of an epigastric splash have to be made with the very greatest care, because it is very easy to be deceived by it. It is true that you get a splash over a dilated stomach, but you may get it also over a displaced stomach, or over the colon, or even over a normal stomach, one which is neither displaced nor dilated, provided the abdominal wall be sufficiently thin. So one should never trust to the presence of a splash alone in the diagnosis of dilatation, but percuss out the limits of the organ. In other words, dilatation of the stomach has to be diagnosed by percussion and not by palpation.

Symptoms of Dilatation.

With regard to the *symptoms* of dilatation of the stomach, I have already said that there is none which is absolutely characteristic. Still, there are some which are certainly common, some which should at least cause suspicion that dilatation is present.

In the atonic or primary cases, the chief symptoms of which the patient complains are a feeling of weight and of flatulence. In atonic cases there is rarely vomiting, and there is very rarely any actual pain. The patient's chief complaints, indeed, may not be of his stomach at all, but are often of what one might describe as the neurasthenic order, exhaustion on bodily exertion, inability for mental work, wasting, and so on, and there may be comparatively little to direct one's attention to the state of the stomach in the history which he relates.

In a case of secondary or obstructive dilatation the symptoms are usually much more pronounced, and one gets the form of vomiting which is most characteristic of this condition, namely nocturnal vomiting. The vomited material is often enormous in amount, fermenting, and exhibits on examination the presence of sarcinæ and yeasts. In such a case the patient will often complain of offensive or acid eructations. Pain of a cramping sort is also experienced in many cases.

It would be going too far to consider to-day the diagnosis of the different causes of obstructive dilatation, and, indeed, this is often by no means an easy matter. In particular one will have difficulty

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in deciding between malignant and benign stenosis. Unfortunately, there is, as you know, no certain method of making an early diagnosis of malignant disease in the stomach. Still, there are one or two points which should lead one in cases of this sort to at least a reasonable degree of certainty in the matter. In the first place, a patient suffering from malignant obstruction usually gives a comparatively short history; he will tell you of an illness lasting only a few months perhaps. Previously to that he will not have suffered in any way from the stomach at all. A patient with benign stenosis, on the other hand, almost always gives a history of prolonged dyspepsia, which has very likely been of a painful character, and he will often tell you that he has on one or more occasions vomited blood. Apart, too, from the difference in the length of the history given in the two cases, you will find that there is more cachexia in the malignant cases than in the benign. But the really difficult cases are those in which the carcinoma has begun in an ulcer, which probably happens much more frequently than we have hitherto supposed. If the patient has had a long-standing ulcer of the stomach situated near the pylorus, and if in that ulcer carcinoma has developed, it may be exceedingly difficult to say that the carcinoma has started, because you will find that examination of the stomach contents in such a case gives you but little assistance. When you examine by means of a test meal in a case of malignant disease, you will find that the free hydrochloric acid is greatly diminished in quantity, or even absent, whereas in a case of benign stenosis hydrochloric acid is in excess. Unfortunately, however, in a case in which carcinoma has begun in an ulcer you will also find plenty of free hydrochloric acid. I have had several cases in which one has found this, and often in comparatively young subjects. I have had two in the last two or three months in men between thirty-five and thirty-seven years of age, each of whom had a dilated stomach, with a history of old-standing dyspepsia, and plenty of free hydrochloric acid in the contents, so that one supposed them to be cases of benign stenosis, the result of the cicatrisation of an ulcer, and yet at operation they both proved to be cases of malignant disease with enlargement of glands.

There is no way of being sure in such cases, except by exploration, and even then you may be in doubt, for I have known one or two such cases where the surgeon believed at the time of the operation that he was dealing with malignant disease, but in which the subsequent course proved that he must have been mistaken; for the patient made a complete recovery notwithstanding that no attempt was made to remove the thickening at the pylorus, and the patient was alive many years afterwards, which would of course have been impossible if the case had been one of malignant disease. In some cases of chronic ulceration of the stomach there may be enlargement of glands along the lesser curvature, so that even the presence of enlarged glands is not necessarily an indication of malignancy.

Treatment of Dilatation of the Stomach.

I come now to speak of the treatment of dilatation of the stomach, which of course is the most important part of our subject. Here I make a broad distinction again between primary or atonic dilatation and the secondary or obstructive form. In primary dilatation you have to treat the patient and not the stomach, whereas in secondary dilatation you have to treat the stomach more than the patient. The primary dilatations are so much due to neurasthenic conditions, to general want of tone, general feebleness, that one can only hope to be successful in them by treating the patient, as it were, as a whole; whereas the secondary cases, which are due to a mechanical obstacle opposed to the action of the stomach and the passage of its contents onwards, can be dealt with satisfactorily by attending to the stomach condition alone.

Now, as regards the general treatment of the atonic cases, the first step must be to improve, as far as one can, the patient's general nutrition, and in order to effect that it is sometimes necessary to insist upon his eating much more than he has been doing. Many cases of atonic dilatation are kept up by the patient half starving himself, in order to spare the stomach, with the result that he becomes ill-nourished, and the general want of "tone" from which he suffers is perpetuated. All measures must be adopted which tend to improve the general health. Such patients are usually overworked, and one has often to take them away from their business or profession and send them to a bracing locality. A great deal can be done, also, by mental influence.

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They are very dependent on their surroundings and on the cheerfulness or otherwise of their environment. I have known many who were greatly benefited simply by being told that there was no serious dilatation. One sees that often in members of our own profession. I have seen more than one doctor who had the impression that he had a badly dilated stomach which would require operation, and when he was told after examination that he was simply suffering from weakness of the wall of the stomach the mere mental relief from knowing it was only that made an enormous difference at once to the general condition. One can do a great deal with these patients and all neurasthenics by suggestion and encouragement, and that is often a quite important part of the treatment.

With regard to the dietetic treatment of the atonic form of dilatation, I think the only two important things to see to are—that the diet should be dry, and that it should be mainly made up of animal constituents. These patients are not those to whom you should give vegetable foods in any quantity, and they are not the people who can afford to drink with their meals. Most of them find this out sooner or later for themselves, but there is still such a deeply rooted idea that vegetable food is more easily digested than animal food—a proposition which, of course, is not true—that many of them, knowing that they have a weak digestion, try to live on too vegetable or sloppy a diet. They will be benefited by a diet containing a large proportion of animal food and by being forbidden to take liquids when they are taking solids. These are the only dietetic instructions which it is necessary to give these patients, but it is advisable to impress upon them the importance of eating enough, bearing in mind, as I have said, that their tendency is to eat too little.

With regard to the other measures which will help in atonic dilatation, I think there is a great deal to be said for massage, and when I say massage I mean massage which is skilfully applied. The difficulty is to get it skilfully applied, but I think there is no doubt that the Swedish masseurs get the best results in these cases. Then there is the question of electricity. Electricity is often recommended in the primary form of dilatation of the stomach, and it is difficult to make up one's mind as to its effects. But if one is going to use electricity, the sinusoidal current is the best form to employ. It is certainly always worth trying, and may at least influence the mental condition by suggestion. Whether it has any effect in contracting the stomach and increasing its tone and the general vigour of its wall it is difficult to be sure. Certainly it is not an indispensable form of treatment.

So much for the general treatment of the patient, his diet, and the use of massage and electricity. Along with those measures one might consider hydropathic treatment, the effect which can be produced on the stomach by such means as external douching, of course skilfully applied. External douching to the abdominal wall has a tonic effect on the patient as a whole, and is therefore so far beneficial, and if the patient can get it along with the advantages of a bracing climate, massage, and electricity, as he often can nowadays in the modern hydropathic, so much the better for him.

Lastly I want to say something about the use of drugs in the atonic form of dilatation. The effect of drugs in such cases is not very great, and I think one can at most relieve some of the symptoms by them. These patients often suffer from want of appetite, and therefore bitters are useful. Strychnine has the advantage of being a bitter and of having also, possibly, a tonic action on the stomach wall, but I think its effect is a general one on the patient rather than a local one on the stomach itself; one can combine with it a carminative in order to dispel flatulence.

It has often occurred to me that suitable aperients may have an action in promoting contraction of the stomach wall just as they have in stimulating the wall of the large intestine. It is an interesting fact that Seigel's Syrup, which is largely taken for indigestion by the general public, so largely that one cannot help believing that it is sometimes productive of good, owes its activity to aloes, along with certain carminatives. There seems no reason why aloes and cascara should not act on the stomach wall just as on the intestine and stimulate it to empty itself of its contents. At all events, you will certainly find that many of these patients are benefited by taking aperients, not in one dose at night, but in smaller quantities after each meal, and the aperient, whatever it be, may be added to the other remedies already described.

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I would impress upon you, finally, that the treatment of atonic dilatation must be persistent. As a rule such patients have been a long time getting ill and you cannot expect to get them right all at once. And although the condition is less serious than the secondary form of dilatation, the latter is more satisfactory to treat because the problem is a simpler one.

I pass on now to speak of the treatment of the secondary or obstructive form of dilatation. Some of you may say that the treatment of secondary dilatation is surgical, and be inclined to dismiss the matter in that way. But I want to protest, as far as one can, against that view. The treatment of obstructive dilatation is not necessarily surgical unless it be due to malignant disease. The treatment of obstructive dilatation, due either to congenital pyloric spasm or to pyloric spasm occurring in the adult, or to benign stenosis, is not primarily surgical. You can get quite good results by medical means, and not only that, but you can get the stomach to return to its normal dimensions. Even in many cases in which you decide to recommend operation the patient will require medical treatment for a time in order to get him into better condition. If the patient be, as he usually is, considerably reduced in strength, and has been suffering a good deal of pain, the first thing to do is to put him to bed. You may think that sounds a truism, but it is a recommendation which is often not followed. During the last month I have seen two cases of severe degree, one of them malignant, in which the patients had not been put to bed, with the result that the treatment which had been carried out was not as successful as it should have been. Another important thing is to insure a proper supply of fluid to the tissues. In cases of great dilatation of the stomach the patient is constantly vomiting, and the quantity of fluid which he gets rid of is very large, and in such a case many of the symptoms are due to want of water in the tissues. It is pretty certain, for instance, that tetany, which is one of the complications of dilated stomach, is due to this cause and so is part of the wasting. The thirst and much of the discomfort which the patient experiences, too, are due to the fact that he is not getting sufficient water into his body. So quite early in the case one should see to it that this defect is made good by the injection of normal saline into the bowel (one pint) every morning, or morning and evening as may be considered necessary. Under this treatment the amount of urine rises, and as it rises many of the symptoms disappear and the patient becomes more comfortable.

The next point I want to touch on is the dietetic treatment of cases of obstructive dilatation. That is an exceedingly difficult matter. You cannot lay down definite rules about it. One may say, however, that the principle to be observed is that the food should be of such a form and consistency that it passes through the pylorus as easily as possible. That means, of course, that it must be mainly liquid at first—at all events in a bad case. This is an unfortunate necessity, because a liquid diet must be bulky in order to get the proper amount of nutrition from it, and therefore throws weight on the stomach, and tends to aggravate the dilatation, but the importance of making the food so that it easily passes the pylorus outweighs other considerations. As the condition of the patient improves, you may gradually build the diet up by making it more and more solid, until it is very much like that which you give in the atonic or primary form of dilatation.

The next question which arises is, whether you should use antiseptics. I do not think there is any real necessity to use antiseptics in cases of dilated stomach. The only efficient way of promoting asepsis is to wash the stomach out, hence lavage will be an essential part of the treatment. The point is often discussed as to what is the best time of day to perform it. I think the most reasonable time for it is the last thing in the evening, for then you insure to the patient a night with a comparatively empty stomach, and for this reason he will probably sleep better than he would if his stomach were left with some fermenting material in it. It is often helpful to wash out with a one per cent. solution of tannic acid. Nearly all those cases of dilated stomach, especially those with benign stenosis, are aggravated by the irritation produced by over-secretion of gastric juice. That can certainly be checked by using such an astringent solution as tannic acid.

The only medicine which you need employ is one to neutralise the excessive acidity, and the best thing to use is one of the heavy carbonates, such as magnesia or chalk; to these may be added carbonate of bismuth. One may give twenty grains

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each of carbonate of bismuth and carbonate of magnesia and chalk in a cachet as many times a day as is necessary to neutralise the excessive acidity. This combination is particularly useful where the dilatation is not due to actual organic change in the pylorus but to spasmodic constriction of it. As such spasm is largely kept up by the excessive acidity, if you neutralise the latter you cause the spasm to pass off and the dilatation is given a good chance to recover.

Finally, we have to discuss the question of operation in dilatation of the stomach. I have said already that in malignant cases there should be no hesitation in recommending operation. But in the other cases, unless there be a history of recent hæmatemesis, or great pain indicating the presence and probable activity of an ulcer, the treatment which I have described should have a prolonged trial. I was going over my notes of cases of dilated stomach recently, and I found records of several patients with severe dilatation of the stomach from benign causes, probably old ulceration in the majority of cases, who learnt to wash out their own stomachs, and who now, with a lapse in some cases of three years or more—in one nearly five years—are living in perfect health and comfort. I have had the opportunity of examining several of these people during the last two or three days, and one could find hardly any evidence of dilatation at all, the stomach having apparently receded back to its normal dimensions. The history which such patients give is that at first they started using the stomach-tube every night, deriving great benefit from it. By-and-by they were able to get along comfortably by using it every other night, then once a week, and later still only once a fortnight. One of these patients now only uses the tube when he feels uncomfortable, which happens about once in two or three months. I do not pretend to be able to explain how it is that these stomachs recover so well, but I am certain that many patients who are now submitted to the operation of gastro-enterostomy are so submitted unnecessarily. I do not underrate the value of that operation, because it is a procedure which has given brilliant results in selected cases, and there is nothing superior to it. I only wish to say that in many cases I do not think it is as necessary as many people seem to believe. On the other hand, you must not postpone the operation of gastro-enterostomy too long; it would not be fair to the surgeon or to the patient to wait until the mucous membrane of the stomach had undergone degeneration from gastritis. The end of gastritis is atrophy of the mucous membrane, and the patient who has that is in an unfortunate position. If washing out has had a fair trial, and the patient is still unrelieved, then I think, unless there be a strong contra-indication, he ought to be allowed to have the benefit of operation.

In primary dilatation, on the other hand, operation should be avoided. I have seen two cases in which it was performed, and in neither had it done the least good. Even washing out is rarely, if ever, called for in primary dilatation. Lavage is a procedure which has been used too freely; the only real indication for it is stagnation of the contents, with fermentation.

September 3rd, 1906.

Sleep and the Hypophysis.—Salmon hazards the suggestion that physiological sleep may be essentially due to some internal secretion of the pituitary body, a hypothesis which, he thinks, harmonises perfectly with the conception of the trophic and antitoxic function of the hypophysis in relation to the nerve-centres. He presents fifteen arguments to sustain this assumption, eight based on the somnolence observed in acromegaly and other affections in which there is hypertrophy of the hypophysis, and in conditions in which the secretions—including those of the hypophysis—are augmented, as in mild pilocarpin intoxication, also in all affections liable to induce hyperæmia in the hypophysis, as in drunkenness, epilepsy, injuries to the skull, etc. On the other hand, insomnia is noted when the hypophysis is destroyed or is undergoing degeneration, also in exophthalmic goitre, in which this gland has sometimes been found exceptionally small and hard, also in old age and in inanition, when the blood-pressure is reduced, and in neurasthenia, which is characterised by low vascular tone and general hyposecretion. Sleeplessness is also noted in intoxication from atropin, which has an inhibiting action on the secretions, and, lastly, under the influence of emotions. This assumption explains also the disturbances in sleep in diseases such as diabetes, in which the glands with an internal secretion are all more or less affected. It also throws light on the disturbances in sleep accompanying changes in the sexual organs and in the nose, and in pregnancy, etc.—*Journ. A. M. A.*, vol. xlvii, No. 5.

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SOME REMARKS
ON
DEFORMITIES OF THE ARCH OF THE FOOT AND OF THE TOES.*

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(*Concluded from p.* 315.)

Deformities of the Toes.

I now propose to consider the various deformities of the big toe, such as hallux valgus, flexus, etc. As in the former lecture, the question of deformities of the infantile and other paralyses will not be considered.

The causation of hallux valgus is given in an excellent text-book under four headings—mechanical, muscular, ligamentous, and arthritic. Of these four two stand out in great prominence—namely the mechanical and the arthritic. There is no disputing the fact that the mechanical shape of the boots has a great deal to do with the production of the deformity of hallux valgus; and there is equally no doubt that all cases cannot be accounted for in this way. The mechanical causation of hallux valgus has been made so prominent by teachers of surgery, that the question of the changes which the boots cause in the bones of the great toe and its metatarso-phalangeal joint have become obscured.

There is no need to recall to mind the enormous importance of the ball of the big toe in walking and running. It will suffice to point out that the head of the first metatarsal is subject to infinite changes of pressure and innumerable small injuries in the course of a day's work. The result should be to bring about osteo-arthritic changes in the joint. Yet if "great toe joints" are examined post mortem, such changes will not be found nearly so often as would be expected! But a moment's thought readily explains this. In walking, the os calcis in the heel bears greater and quite as numerous traumatisms as does the big toe joint; yet, osteoarthritis of the calcaneo-astragaloid joint is not so very common. In other words, the second problem, enunciated about the heel, is very like the first, which concerned the great toe. Osteoarthritis con-

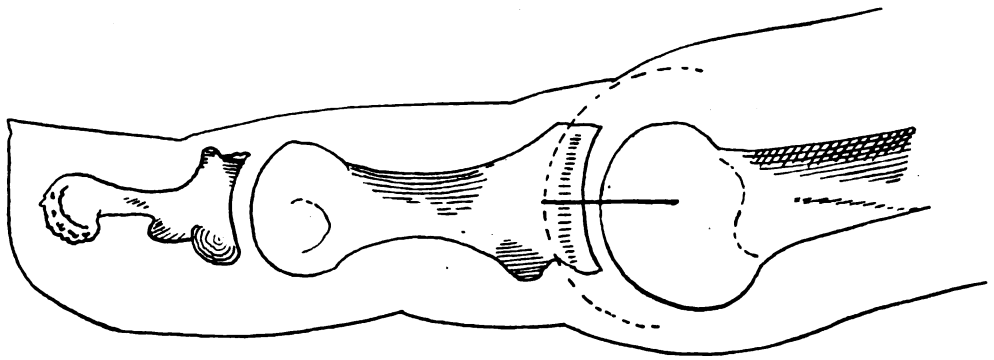

Fig. 8.—Diagram of the normal big toe joint. The dotted line indicates the arc of a circle described in movements of the toe; the black line is the lateral ligament and radius of that arc.

sists of two parts, as its name suggests—one of the bone and one of the joint. Any bony point which has to bear frequently applied pressure or pull becomes thickened, as can be seen in the tuber ischii, the calcaneum, the trochanter, or in any muscular attachment. In the case of the heel in our second problem, the bone becomes much thickened; in pathological parlance, it might be said to become enlarged and eburnated. No such changes are described in the head of the first metatarsal bone. But they must and do occur. But these bony changes take place without there being necessarily any changes in the joint. So that it may be said that the "osteo" changes can occur without any "arthritic" changes. What effect will this thickening have on the big toe? It will (1) apparently displace the articular surface of the metatarsal bone dorsally, (2) cause enlargement of the under part of the

* Delivered at St. Thomas's Hospital.

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head of the metatarsal bone, and (3) it will limit the downward movements of the toe at the metatarso-phalanjeal joints. The first two are obvious, and require little or no remark. The last requires a word or two of explanation. In the perfect joint, as seen in a young child, the first phalanx of the big toe moves on the metatarsal bone in the arc of a circle whose radius is represented by the length of the lateral ligaments (see Fig. 8). The curve of the articular surface is roughly that of a circle. When the lower part of the head of the metatarsal becomes enlarged the curve is no longer that of a circle. So that the lateral ligaments by becoming tight prevent the phalanx from riding over the bump. The enlargement of the under part of the head of the metatarsal possesses an important clinical bearing. By means of the limitation of plantar flexion of the toe, that structure slowly and surely gets displaced and flexed on to the dorsum of the head of the metatarsal, a position I would designate with the name of *hallux extensus* (Fig. 9).

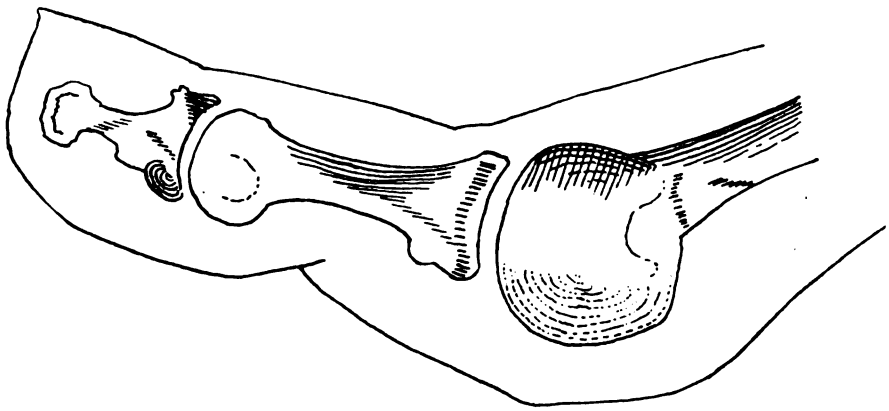

FIG. 9.—Hallux extensus with enlargement of under part of the head of the metatarsal.

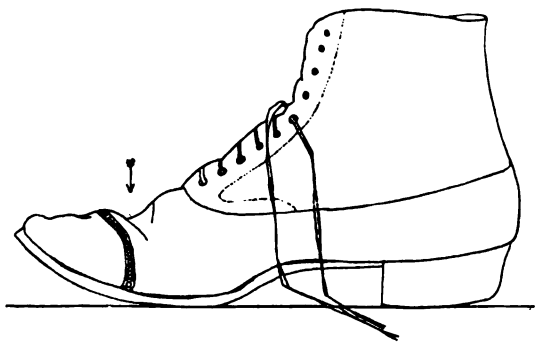

FIG. 10.—The boot of a patient with pes cavus, showing the curling up of the toe and the transverse creasing.

Those who have high insteps—*i. e.* a slight pes cavus—use the ball of their big toe a great deal more than do flatter footed people, with the result that the bony changes in the plantar part of the head of the first metatarsal bone are greater, and the toe is curled backwards towards the dorsum of the foot. Hence the transverse crease across the bases of the toes in the boots of those who have high insteps. This becomes more and more marked in severer examples of pes cavus.

Hallux extensus is flexion of the first metatarsal joint towards the dorsum of the foot, and is associated with limitation or absence of plantar flexion of the great toe, enlargement of the plantar part of the head of the metatarsal bone, and increase of the natural or normal arch of the foot —pes cavus. *Hallux flexus* is the opposite of hallux extensus, as its name implies, the big toe being plantar flexed towards the sole of the foot. *Hallux rigidus* is a condition in which the big toe is in a position halfway between flexion and extension; it is painful and cannot be dorsally flexed, whilst it can be in the plantar direction. If this deformity progresses the big toe slowly becomes bent more and more into the sole, becoming hallux flexus. In this lies the germ of its pathology. Hallux rigidus is associated with the abducted or resting position of the foot, frequently with definite flat foot. These people walk with toes turned out, their weight being borne by the outer side of heel and a callosity under the third toe The result is that the ball of the big toe is jammed tightly against the "upper" of the boot at every step. In consequence, it is the upper or dorsal part of the head of the first metatarsal bone which

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becomes enlarged, exactly the opposite to what happens in hallux extensus. In hallux rigidus the articular surface of the head of metatarsal bone is displaced in the plantar direction; the head of the metatarsal bone is enlarged in its dorsal part; and lastly, the movement of dorsal flexion (extension) of the big toe is limited, because the enlargement

My explanation also solves the question of hallux rigidus and hallux flexus as those names are ordinarily understood. But if the deformities of the foot are viewed as a whole, it is seen that the condition of hallux rigidus I have described is the *hallux rigidus of the abducted or resting position* of the foot. Is there a corresponding deformity

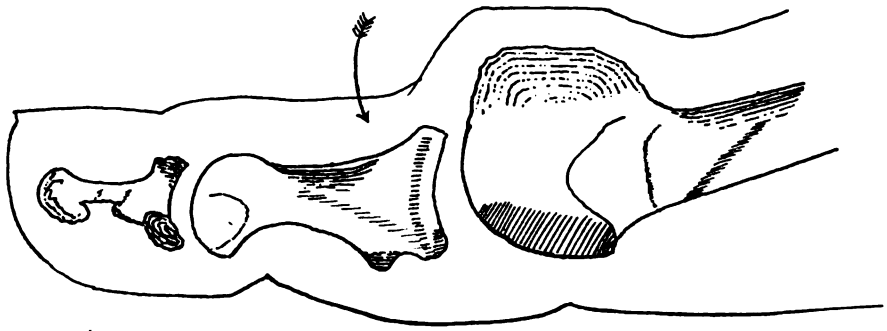

FIG. 11.—Hallux rigidus. Dorsal enlargement of head of metatarsal.

alters the curvature of the articular surface of the head of the bone, and the lateral ligaments, becoming tight, prevent dorsal flexion of the toe. If the dorsal part of the head of the metatarsal bone becomes more enlarged, the toe will be bent more and more towards the sole of the foot—that is to say, bent into the position of hallux flexus.

What I have just said explains the production

for the adducted or active position of the foot? Yes, certainly; it is so common as to represent almost the normal great toe, and as it gives rise to no trouble it has never been described. In it the great toe cannot be flexed in the plantar direction, whilst it can be flexed dorsally—that is to say, the precisely opposite condition to that of the hallux rigidus of the abducted foot.

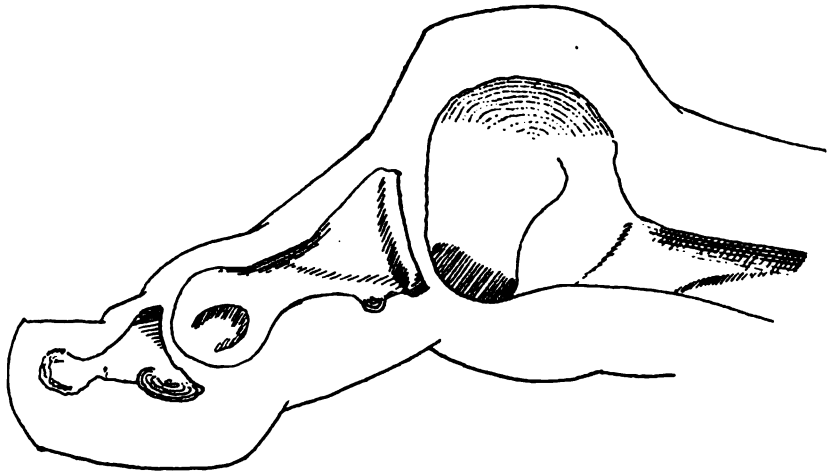

FIG. 12.—Hallux flexus. Exaggeration of the condition in Fig. 11.

and associations of hallux rigidus and hallux flexus, how dorsal flexion is abolished, and how the first deformity may pass into the second.

The presence of the dorsal enlargement of the head of the metatarsal bone can be easily recognised clinically.

Now, it will be seen that hallux flexus is associated with the abducted, flat, or resting position of the foot; that hallux extensus is associated with the adducted, arched, or active position of the foot; and each has a condition of the big toe, for which the present-day name is hallux rigidus, which

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represents the corresponding deformity in a minor degree. Thus, there are two types of hallux rigidus, one leading to flexion and the other to extension of the great toe.

Further, if one examines the movements of a big toe suffering from hallux valgus, it will be seen that this deformity also has a condition of hallux rigidus associated with it.

The most common deformity of the big toe is undoubtedly *hallux valgus*. This deformity is very common among women, and is doubtless attributable in great part to the median pointing of their boots and shoes. When speaking of pes planus it was pointed out that the high heels of women's boots prevented them getting the ordinary recognised flat foot by retaining some of the natural arch of the instep. The foot was, however, in the flat, resting, or abducted position. When talking of hallux rigidus,* it was pointed out that this deformity arose from the flat or abducted position of the feet. Yet clinical experience tells us that it is hallux valgus and not hallux rigidus alone which is found in ladies' feet. So that it is not unnatural to suppose that hallux valgus is in some way the female counterpart of the hallux rigidus of males.

If the heads of the metatarsals are examined, it is noticed that they are enlarged on the dorsum in hallux rigidus and on the inner side in hallux valgus. The difference in the situation of this bony outgrowth is easily explained by the different points of pressure of the "upper" of the boot whilst walking with abducted feet, in one case with the heel on the ground and in the other with the heel artificially raised, as in a lady's boot. In the former case it is the dorsum of the head of the first metatarsal bone which bears most pressure, and in the latter it is the inner side, each developing the corresponding bony outgrowth. Hence a woman with abducted feet develops hallux valgus and a man with abducted feet hallux rigidus.

It is a very common clinical observation, which is easily made, that it is frequent to see big toes in a position intermediate between hallux rigidus and hallux valgus; a good deal less frequent is the position between hallux flexus and hallux valgus.

Besides the causation due to the development of a bony deformation of the head of the metatarsal bone, hallux valgus also has another. The big toe becomes abducted—*i. e.* moved into a position of slight hallux valgus, as part and parcel of the abduction of the foot.

Hallux valgus, when produced as a primary deformity as the result of mesially pointed boots, has a very deleterious effect upon the future use of the foot. Even though people may have a good active arched instep, should they develop a primary hallux valgus, the abducted big toe prevents the foot assuming the position of activity, and the arch of the instep slowly and steadily flattens, the foot becoming abducted. A primary hallux valgus in young adults is, in consequence, a very serious thing for their future activity and health. Treatment to rectify the deformity and the use of proper boots alone can restore their feet to the natural condition, and if this moment is allowed to pass by irreparable harm is done to the patient.

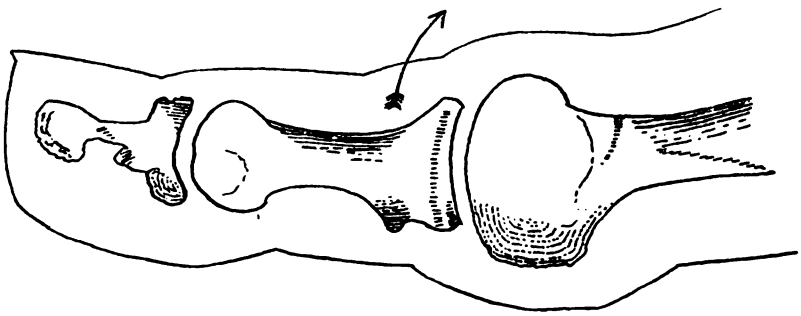

FIG. 13.—The hallux rigidus associated with hallux extensus. Compare with Fig. 11 in which the deformity of the metatarsal is on the dorsum whilst here it is on the under part.

Hallux varus is a name which is not in common use and conjures up no familiar picture. As a deformity it is far more frequently seen than recognised because the adducted big toe, which constitutes hallux varus, is part and parcel of the exaggerated adducted position of activity of the

* The hallux rigidus here referred to is the one which is recognised—*i. e.* that of the abducted position.

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foot, called a high instep or pes cavus. It is a deformity due to the adducted position being made permanent. It differs from other deformities of the hallux in not being due to the development of a bony growth on one or other side of the head of the metatarsal. It is useful to recall the deformity as it completes the tale of deformities of the big toe—hallux rigidus, hallux flexus, hallux extensus, hallux valgus, and hallux varus.

Hammer toe.—There now remains a series of deformities to be considered, of which hammer toe is the one which has earned for itself a clinical reputation for demanding attention.

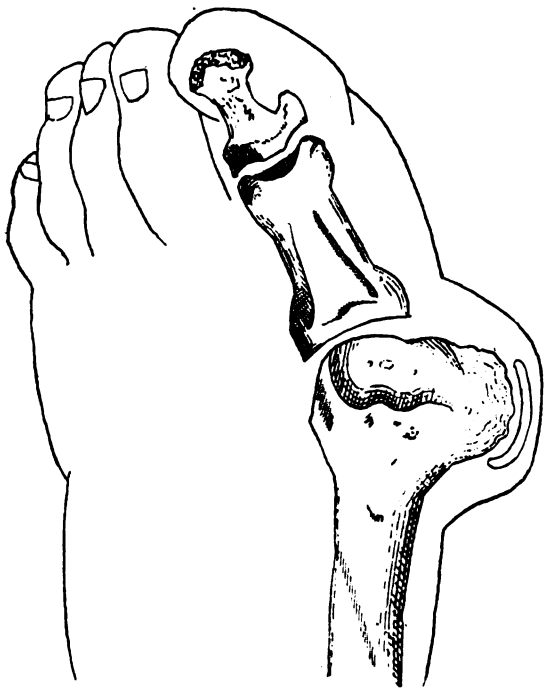

FIG. 14.—Hallux valgus.

First of all, the toes must be examined with the foot arched in the position of activity and when flattened in the position of rest. If the naked foot of an athlete is examined when about to start to sprint, it will be noticed that besides the various points in the position of activity already mentioned, the big toe is in the position of hallux extensus, and all the other four toes are bent into similar positions. They are dorsally flexed (extended) at the metatarso-phalangeal joints, plantar flexed at the first, and dorsally flexed at the second interphalangeal joint. Thus, there is a definite position of the toes associated with the position of activity of the foot, which position, it may be noted, is the same as that in the deformity called hammer toe. In hammer-toe the prominences of the flexed joints through rubbing against the boots are covered by corns and bursæ, which not infrequently become inflamed. Were it not for these corns and bursæ no complaint of the condition would be made.

As all the toes in activity of the foot are in the position of the deformity of hammer-toes, *digiti extensi*, it is a question as to why it is the second toe in which the position becomes so much exaggerated that it develops corns, etc., whilst the other three are not painful. The second toe becomes more bent up for two reasons: firstly, because, owing to its great length and prominence, it has to be more bent in order to accommodate itself; secondly, owing to the presence of some degree of hallux valgus, the big toe displaces and bends up the second toe still more.

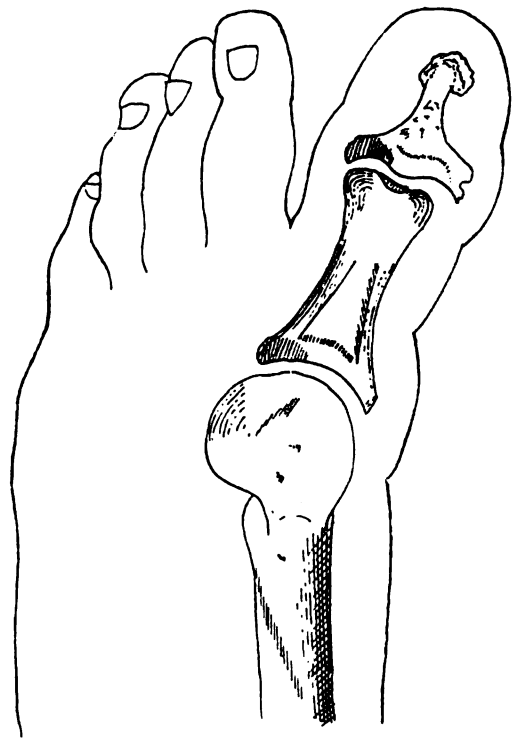

FIG. 15.—Hallux varus.

To recapitulate, the position of the foot in activity is associated with a position of the toes, *digiti extensi*; in the case of the second toe this position is apt to be exaggerated, causing pain

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and lameness, when the condition is called hammer-toe.

The further problem is the position of the toes of the foot when flattened and at rest. The toes are straighter and tend to continue the lines of their metatarsal bones. Moreover, they lose, or have already lost, their capacity of dorsal flexion (extension). The position of the big toe is similar and is called hallux rigidus. Therefore, the toes had better be termed *digiti rigidi*. Further, as hallux flexus can follow on hallux rigidus, so can the rigid toes become plantar flexed, becoming *digiti flexi*. With arched insteps, the second toe is apt to become bent up and painful—hammer-toe. With flatter feet, the second toe is projected straight out, lying either above or below the "crowded-in" big toe, according to its position.

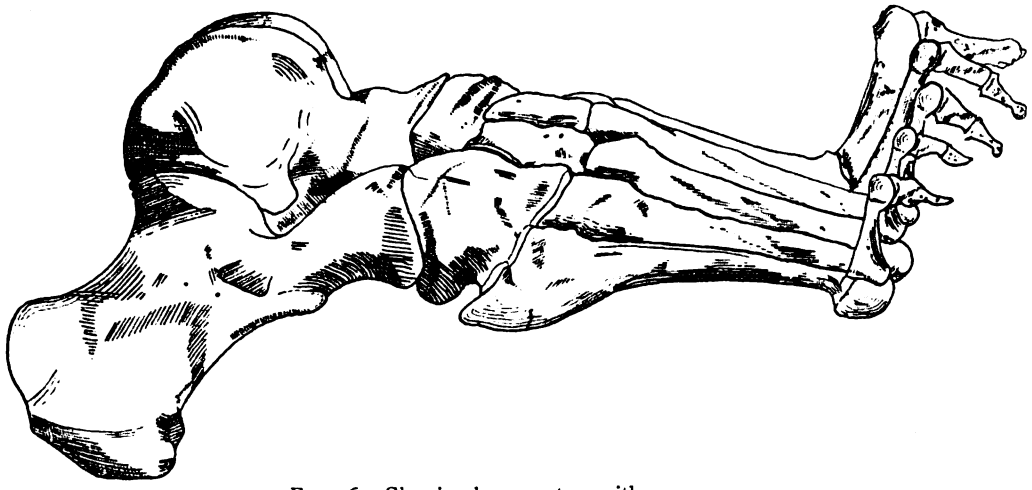

FIG. 16.—Showing hammer toes with pes cavus.

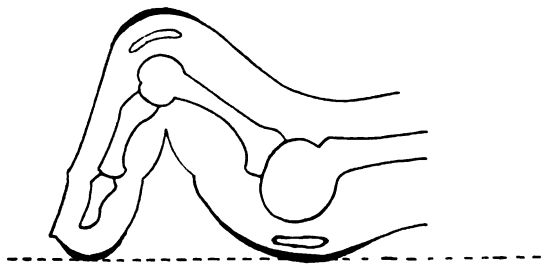

FIG. 17.—Hammer-toe, showing corns and bursæ.

You will remember that there are two kinds of limitation of movement of the great toe which can be called hallux rigidus, one associated with hallux flexus and the other with hallux extensus. Similarly two kinds of *digiti rigidi* can be recognised, one, tending to *digiti flexi*, in which the toes cannot be dorsally flexed (extended); the second, tending to *digiti extensi*, in which the toes cannot be plantar flexed. The former is associated with the position of rest and the latter with that of activity of the foot.

Pes cavus is the deformity resulting from the correction of the inversion (varus) of the active and adducted position of the foot; typically it is associated with equinus.

Pes planus is an exaggeration of the eversion (valgus) of the resting and abducted position of the foot; typically it is associated with calcaneus.

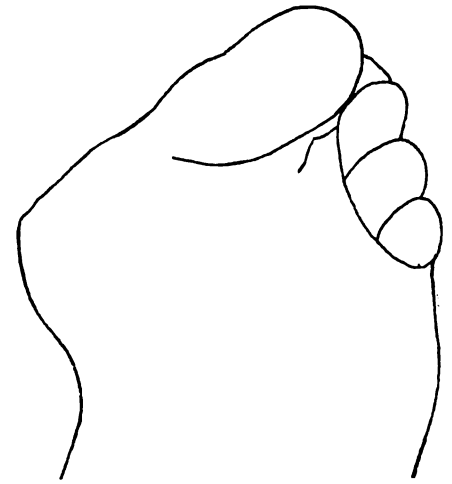

FIG. 18.—Example of digitus flexus (second toe) seen from the sole of the foot.

Hallux extensus and *digiti extensi* (including

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hammer toe) are deformities associated with the arched and active position of the foot.

Hallux flexus and *digiti flexi* are deformities associated with the flat and resting position of the foot.

Of *hallux rigidus* there are two chief types, one tending to hallux extensus and one to hallux flexus; there is a third type associated with hallux valgus.

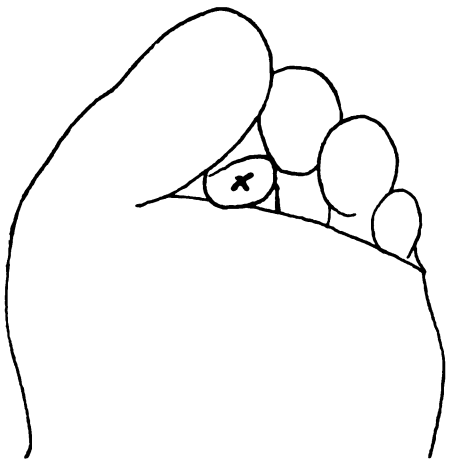

FIG. 19.—Example of hammer-toe (digitus extensus x) seen from the sole of the foot.

Hallux varus is an incident in the adduction of the foot into the arched and active position.

Thus there are many dispositions of the joints of the foot which are perfectly typical of the active or resting attitudes; some, on account of their drawing attention to themselves, such as hallux flexus, are called deformities, whilst others, which do not call attention to themselves, are not called deformities, such as hallux extensus.

September 3rd, 1906.

Gallop Rhythm.—Müller concluded a post-graduate lecture with the remark that the prognostic significance of gallop rhythm is not so grave as some are inclined to believe. In case of goitre, of cardiac neuroses, and even in typhoid fever, the phenomenon does not indicate danger. Even in a slowly progressing contracted kidney, as also in many heart affections, such as those in alcoholics, for instance, he has observed that patients remain in comparatively good condition for many years after the appearance of the gallop rhythm. However, it repays study and is frequently an indication for energetic treatment, especially for digitalis—*Journ. A. M. A.*, vol. xlvii, No. 5.

PERICARDITIS IN CHILDREN.

By H. W. SYERS, M.A., M.D.Cantab.,
Physician to the Great Northen Central Hospital.

IT certainly appears that pericarditis is much less commonly observed than formerly as a complication of acute rheumatism. Twenty years ago nothing was more usual than for the malady to be met with in combination with the articular manifestations of rheumatic fever. At the present day it seems to me that pericarditis is a comparatively rare accompaniment of acute rheumatism, while valvular disease of the heart is just as commonly, or, indeed, more frequently, met with.

Further, when pericarditis does appear in the course of acute rheumatism in children at the present time, it is generally in those cases which are characterised by little or no joint affection, the articulations practically escaping, and the utmost that is complained of is some aching in the joints or limbs, and "growing pains."

Yet when pericarditis does appear it would not seem to have lost any of its virulence, for it appears to be more aggressive, more assertive, than hitherto. Not seldom, too, and in spite of the greatest care, when the attack has apparently come to an end, a second supervenes, and the whole of the previous incidents are repeated. Several such attacks may ensue, and these may be separated from each other by an interval of a few days only, or it may be a few weeks. This being so, it is not difficult to understand how it is that entire adhesion of the two surfaces of the pericardium rapidly ensues and also that the muscular structure of the organ undergoes most serious degenerative changes, ending in too many cases in dilatation of heart cavities, which eventually leads to a fatal termination.

I think, then, that it may be asserted with considerable confidence that pericarditis is nothing like so usual a concomitant of acute rheumatism as was formerly the case, but that when it does occur it is more acute, more tenacious and persistent, being attended with greater risk than formerly, both as regards the complete obliteration of the pericardial cavity and of subsequent degeneration of the muscular structure of the heart.

An important physical sign of acute pericarditis in children concerns the relationship of the distended pericardial sac to the lower lobe of the left

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lung. It is not very unusual for the distended pericardium to press on the left lung in such a way as to encroach on the vesicular structure of the lower lobe of the organ; to such an extent may this pressure be carried that a suspicion of pneumonic consolidation of the left lower lobe may arise in the mind of the observer. Quite recently I have had an opportunity of watching a case of this description, the patient being a girl, æt. 9 years. There were no joint symptoms, but previous typical attacks of acute rheumatism had occurred in which the endocardium had been damaged, as there was both aortic and mitral disease. There was very marked outpouring of fluid within the pericardium, and when the effusion was at its height the percussion note over the whole left lower lobe became dull. Over the dull area auscultation revealed the presence of marked bronchial breathing, which extended to the left lateral region. Now, as the temperature was considerably raised, and as there was marked dyspnœa the condition closely resembled that characteristic of consolidation of the left lower lobe. But that most important physical sign, the presence of an altered pulse-respiration ratio, was wanting, and it was perfectly clear that the real explanation was that pressure on the vesicular structure of the left lower lobe caused loss of percussion resonance and also led to the transmission of the bronchial breath sounds from the root of the left lung, which replaced the ordinary vesicular murmur in normal conditions audible over the pulmonary base.

Just in proportion to the rapidity of the absorption of the pericardial fluid did the physical signs at the base of the left lung disappear, until with the entire absorption of the pericardial effusion the condition at the left base became once more normal. But in a few days the temperature once more rose, and again pericarditis with effusion appeared. The amount of fluid poured out on this second occasion was, however, considerably less than before, doubtless owing to the fact that adhesions of some tenacity rendered impossible a recurrence of the extreme distension of the pericardium previously noticed. The phenomena just detailed again occurred at the left base, and once more they gradually disappeared, contemporaneously with the absorption of the pericardial effusion.

It is important to bear in mind the possibility of pressure upon the lower left lobe by a pericardial effusion giving rise to physical signs closely resembling those of consolidation due to pneumonia; in this way the error of attributing these signs to the presence of pulmonary inflammation will not be fallen into. I am inclined to think that this mistake is more often made than is generally supposed.

As a rule the diagnosis of pericarditis from the presence of friction sound is easily made, but not in all cases is it possible to avoid error. Mistakes are liable to be made in the early stages of the malady, for then a friction sound and a double aortic murmur are apt to be confounded. I recall a case of this kind in a boy of fourteen, seen in the out-patients' room; there was high fever, pain in the precordial region, and great dyspnœa. Over the whole precordial region a double murmur was audible, exactly resembling a friction sound. At the post-mortem examination it was found that the aortic valves were the seat of extensive calcareous deformity by which the aortic orifice was greatly narrowed, and that the valves were affected with malignant endocarditis of quite recent origin. The pericardium was perfectly normal. It is true that in this case the apex beat was clearly perceptible, and that hypertrophy of the left ventricle was present, but it was assumed that pericarditis existed in its very earliest stage, sufficient time not having elapsed for effusion to occur. The case was obviously one of malignant endocarditis, involving an old rheumatic lesion.

In my experience mistakes similar to this are not uncommonly made, the error invariably being to interpret as due to pericardial friction sounds which are in reality caused by structural changes and incompetence at the aortic valves.

Concerning the loudness of aortic murmurs, both in children and adults, I have been struck by the fact that not infrequently the loudest, most harsh, and rasping murmurs are those which are due to the presence of very fine, soft granulations located on the edges of otherwise undamaged aortic valves; the latter are, in these cases, quite undeformed and free from all shrinking and thickening. The latter changes may, of course, be attended with the production of loud, harsh murmurs, but in no degree more so than when such alterations are entirely absent, nothing more than soft, recently-formed granulations being present. Doubtless the valve which, with the exception of the deposit on its free

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margin, is essentially healthy, forms a more efficient reed than does the one which is more or less thickened and rigid, and therefore much less movable and capable of vibration. Hence probably the peculiarities of the murmurs elicited when the morbid changes are confined to the presence of granulations at the point of contact of the aortic cusps, and hence, also, the musical character which is occasionally noticeable in connection with these murmurs.

A very wide, and by no means fully explored, field is that relating to the vagaries of heart-action which are observed in chorea, and therefore in children predisposed to suffer from acute rheumatism. I do not here refer to the well-known systolic murmurs so often audible in these children. No doubt in most cases these murmurs are of functional origin, though it is not at all easy to say in a given case whether or no organic disease is present. But it is not at all uncommon when auscultating choreic children, in whom the movements are very slight, perhaps limited to twitching of the facial muscles, to find alteration of the first or second sound, or of both, sometimes combined with an organic murmur.

In a girl, æt. 12 years, there was a loud organic mitral systolic murmur, but the state of the heart in other respects was scarcely the same for two consecutive days, this unstable condition lasting for several weeks, during which choreic twitchings of the facial muscles were more or less constantly present. The child had suffered from acute rheumatism, but no joint-pains, except some slight discomfort in the left shoulder, were complained of during the persistence of the abnormal action of the heart. This abnormality consisted in the presence of a marked reduplication of the first sound, which gave rise to a cantering heart-action strongly suggesting the earliest stage of pericarditis. The advent of this malady seemed the more probable inasmuch as the temperature was somewhat raised. But in a day or two the condition changed, the first sound became whole, still accompanied with the systolic murmur, but the second sound was now loudly reduplicated. After a few days another change occurred—a presystolic murmur with a decided thrill, perceptible on palpation, developed. This murmur came and went, its presence alternating with that of a reduplicated first-sound. Finally, the whole of these abnormalities disappeared, and the condition became as at first observed—that of mitral regurgitation pure and simple. When last seen the case simply possessed the physical signs of an ordinary organic mitral systolic murmur with complete compensation. There can be no doubt that if seen once only during the presence of the vagaries just described, the heart in this girl would have been regarded as far more seriously affected than was really and in fact the case; there can be no doubt that mitral incompetence was the lesion actually present, and that the other abnormalities were purely functional and the result of chorea. It is not often that the opportunity occurs of following a case of this description from the commencement to the end of these functional manifestations, and it seems to me that such curious cardiac abnormalities deserve detailed study and that their occurrence should be anticipated in cases of chorea and rheumatism similar to that of which I have given the history.

September 3rd, 1906.

Fatigue.—Lee has shown that physiologists are generally agreed as to the facts of muscle fatigue, but the condition of the peripheral nerves and nerve-centres is still a subject of dispute. The recent results would indicate that the muscular system tires before the nerve-centres. They also throw a certain measure of doubt on the supposed proofs of central fatigue. A physiological explanation of the facts of mental fatigue is not possible in the present state of research. There is no denying the fact of psychical fatigue, but it is unknown whether it is of central or peripheral origin. Metabolic products, such as sarcolactic acid, monopotassium phosphate, and carbon dioxide, have a toxic depressant action, especially on the muscular system, and the sensation of fatigue is in large part the psychic manifestation of this depression. Little is known as to the production of fatigue substances in the central nervous system, but the action of such substances is not confined to the tissues in which they arise; excessive activity of one tissue can cause fatigue of others. The effects of acid intoxication are similar to fatigue phenomena. There has been very little serious endeavour to provide specific antidotes for fatigue considering its importance. Alcohol in small amounts seems to have a favourable action, but it is followed by bad after-effects and cannot be classed as a valuable antidote. Both scientific and unscientific experiences have shown the value of sugar as a partial restorer of working power. An alkali such as sodium bicarbonate may not be without value—*Medicine*, vol. xii, No. 7.

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A CLINICAL LECTURE ON URTICARIA.

Delivered at Charing Cross Hospital.

By JAMES GALLOWAY, M.D., F.R.C.P.

GENTLEMEN,—The subject noted for to-day's lecture in this Session's course is, I see, "Erythema exudativum and Urticaria." The subject is of more than ordinary interest, not only from a purely dermatological aspect, but on account of the numerous relations of these affections to general diseases, especially, perhaps, in the case of Erythema exudativum. In the last course of lectures we considered especially the subject of Erythema exudativum, so I think it is fair to put urticaria first in our lecture to-day.

I will first draw your attention to this patient. He has come to the hospital complaining of troublesome irritation of the skin, so considerable as to prevent him sleeping at night. He says that in the evening numerous whitish blains appear on different parts of the body, accompanied by severe itching, so that he cannot prevent himself from rubbing his skin.

On looking at the patient you will see at present almost nothing, but if you look very carefully you will find about the shoulders and the waist where his clothes have been pressing slightly raised lines of skin, whiter than the normal colour. They seem to follow the lines of pressure and foldings of his garments. I will now take this penholder and draw the butt of it in various directions across the skin of his back. The first result is, as you observe, whitened streaks owing to the temporary absence of blood produced by the pressure. You will now notice how very rapidly the lines I have drawn become red and that the pink stripes become crimson in marked contrast to the normal colour. In the centre of the crimson stripes you will now

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notice that the skin is becoming again white, and that this narrow white line is gradually broadening and raising itself above the surface. In a few minutes you will notice that the white line has become a raised white wheal, that the crimson border is gradually absorbed by the spread of the wheal, and that he has now marks across his back almost as thick as my little finger, well raised above the surface, bordered by only a faint blush of a pink colour. The amount of disturbance appears to be as great as might be produced by lashing an ordinary individual, but it is all the result of the very slight irritation produced in the way you saw.

This very abnormal irritability of the skin is known sometimes by the name of "dermographism," for an obvious reason. To-day, for instance, a lad came to see me after being absent from the hospital for some time, and reminded us of his identity by informing the students that when he attended the hospital previously I used to write his name, "Tom," across his back!

This condition, sometimes known as factitious urticaria, or, in the well-marked cases, dermographism, is useful for us to bear in mind as it is associated with or may even underlie many of the cases of urticaria and urticarial-like eruptions which come under our notice, and some physicians pay much attention to it as of prognostic significance. Dr. McMurray, of Sydney, N. S. W., especially has drawn attention to this matter, and suggests methods for estimating the degree of skin irritability indicated by this phenomenon.

Urticaria, then, is a disease of the skin in which the primary lesions are known as wheals or pomphi, the latter word coming from a Greek source, signifying a blister, and making its appearance in other terms such as pompholyx and pemphigus. In true urticaria the lesions do not go on to actual vesication; in the great majority of cases they stop short with the œdema of the skin which you have just seen in this case of factitious urticaria, and after a longer or shorter period die away. It will simplify what I have to say if you will please to bear in mind that there are two sets of causes capable of producing urticaria. One of them is of external origin, the other arises from within the body. So far as the appearance of the lesions on the skin is concerned there is little difference from whatever source the disease may originate, but bearing this in mind, you will have a clue to follow in investigating the origin of the disease in any case.

This patient comes before us with a not uncommon history. He is a man aged about 50 years, and tells us that for four or five weeks he has been suffering from much irritability of the skin, producing pruritus, so that he is unable to sleep at night. Coincident with this itching there appear on the skin bumps which he says are exactly like those which might be caused by a nettle sting. When he came before us a few minutes ago in the out-patient department, he had on the lower part of the body linear lesions of the type I have described to you, which have now disappeared. These lesions were of the nature of factitious urticaria, and were produced by the creases and pressure of his clothing, and you will see that his skin is highly irritable because I am able to produce the factitious urticaria which has been already demonstrated. What is the cause, then, of this man's discomfort? He appears to be in fairly good health, and is evidently careful about cleanliness both in person and in clothing. But when you see a case of urticaria lasting for five or six weeks, as it has in this particular patient, you should be very difficult to convince that the patient is really in perfect health. The possibility of external irritants being disposed of, on further investigation we find that the patient suffers from hæmorrhoids, and as a result of these has a good deal of hæmorrhoidal irritation and pruritus. You will notice that the shape of the abdomen suggests that there is a tendency for the abdominal viscera to fall downwards. I cannot make out the liver edge below the costal margin, but he complains of dyspepsia of the type which produces an acid or burning sensation behind the sternum with actual pyrosis. His tongue is unnaturally red, and I think you will agree with me that the stomach is large and probably suffers from chronic distension. You will understand, therefore, that his dyspepsia is caused in all probability by the state of his stomach; that he gives collateral evidence of dyspeptic trouble by the condition of his tongue, and the presence of the hæmorrhoids indicates disturbance of the abdominal viscera of considerable degree. Collecting together this evidence, you will agree, I think, that there can hardly be any doubt of the absorption of badly digested food-products from the intestinal tract, of their imperfect elaboration in the body, and the con-

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sequent unhealthy state both of the blood and the tissues. In such cases the conclusion can hardly be resisted that the urticaria results from this toxæmic process.

The case of a patient such as this may offer difficulties in diagnosis, and the conclusion to which I have come may be open to criticism. When you become house-physicians, however, the most striking cases of urticaria that you will see come to the casualty department. Very frequently a strong labouring man has been indulging too freely in beer and ends off an evening of dissipation by a meal of winkles or such-like delicacy. He comes to the hospital with his face swollen, large lesions of urticaria over the body, and intensely uncomfortable. An emetic and an efficient purgative gets rid of the irritating material from the intestinal tract, and the skin manifestation rapidly disappears. In such a case the causal connection between the absorption of poisonous food-products and the eruption on the skin is too clear to be gainsaid.

There are two other varieties of disease which are sufficiently troublesome and common to deserve special notice. One of them is known sometimes by the name of "chronic urticaria." It is frequently seen in adults and often in persons who have the aspect of being run down in health, and may last for many months. Very frequently the attack comes on in the evening, produces a night of discomfort and restlessness, and occasionally the lesions, instead of vanishing in the ordinary way, leave a small indurated papule, which may prove the source of continued irritation. More than one cause is concerned in the production of this type of the disease, but you will find in nearly every case that the disease has some relation to food or to digestion. It may be an actual article of food which is poisonous, or the imperfect digestion and elaboration of the most simple food, which is ordinarily innocuous, may produce the poison.

I recollect the case of a lady who consulted me with this form of the disease, which had annoyed her for two years. She was thin and of nervous type. During the course of her malady various forms of diet and want of diet had been recommended and tried, but without result. After a very careful examination of the food she was in the habit of taking, it turned out that after dinner, which was usually specially arranged for her, and while the others were having dessert, she had got into the habit of eating an almond, or sometimes only part of an almond. This small fault was the reason of her trouble, for when it was noted and she abstained from eating almonds the urticaria disappeared and she was able to take an ordinary simple diet without any ill result. Many cases of this sort are improved, and sometimes entirely cured, by leaving off the use of alcoholic beverages.

The other variety of the disease, or at any rate a closely allied disease, is what is known as "urticaria of children," or, as it is frequently called, "Lichen urticatus." This condition is very common—you must have seen many cases amongst our out-patients—and it is very difficult to treat. It occurs in early childhood. An infant or a young child is brought, and on examination it is seen that the body, especially the upper parts of the thighs and the lower part of the trunk, is studded with small papules, usually slightly reddened, very often with the tops scratched and covered with a small blood-crust. The mother will frequently inform you that at night the child is covered with small blisters, and if you are fortunate in seeing any of these lesions, you will notice that as a rule they are reddened macules, with usually only a small amount of œdema of the skin. The papules seem to remain as the relics of these erythematous or urticarial lesions and become the source of continued pruritus. Children so affected are obviously very uncomfortable; they usually are making efforts to scratch the affected skin, and are irritable and sleepless at night. There is still a doubt remaining whether the papules which give rise to so much trouble are actually the result of the erythematous spots. Some observers are of opinion that they arise independently of urticarial wheals, and that the disease is closely allied to the exceedingly troublesome condition known as "prurigo."

The cause of this troublesome affection is also somewhat in doubt. You must first make quite certain that there is no source of external irritation. The possible presence of external parasites, such as pediculi, must be carefully inquired into. The cleanliness of the child must then be assured, for irritating eruptions of this type seem to occur in children who are not carefully kept clean and dry. The next, and perhaps the most important, inquiry must be regarding the general health of the child, and especially its diet. Many children suffering

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in this way have the appearance of being rickety, and if this is so special care as to food must be taken. But in many others who show no evidence of rickets the careful selection of the diet is necessary.

There is another condition closely allied to urticaria which I desire to mention to you in order to complete what I have to say to-day, namely what is sometimes called "angioneurotic œdema," or sometimes "giant urticaria." In this state there occur usually in the adult enormous wheals, to which the ordinary lesions of urticaria are but pigmies. They may occur on almost any part of the body. A large area, such as the side of the face, may be involved, the skin swollen, almost translucent, and the features completely masked. This state comes on suddenly, lasts for a short period, and then usually disappears, leaving no trace. Its aspect is so striking as to give rise to very serious alarm as well as discomfort. A few of these cases appear to become chronic. One young man recently attending the hospital suffered from this type of the disease; his lips were constantly swollen, especially on one side, the face being also affected on that side. Without any reason sudden attacks of swelling came on, producing great discomfort.

I draw your attention to this peculiar state in order that you should recognise it when you see it and remember that though resembling ordinary urticaria in the characters of its lesions, it is not so clearly connected with food and with errors of digestion and the assimilation of food.

The means of treatment of urticaria are important to understand clearly, so that they may be applied with effect. The patients see the lesions and feel the discomfort; they expect relief and are in the best position for appreciating success in treatment. In reference to the acute cases the treatment has already been indicated. If the intestinal canal is emptied of its poisonous contents, the absorption of the poisonous material ceases and the skin manifestations gradually diminish. Rest and the use of some of the sedative local applications to be mentioned will produce comfort and complete the cure. It is in the chronic cases that the difficulties as to treatment present themselves. Care as to diet is the first great point requiring attention; in many cases this is all that may be neccessary. It is sometimes thought that patients who suffer from the train of errors in assimilation which give rise to gouty symptoms are also liable to suffer from urticaria, and this is likely to be true, but some of the most resistant cases of chronic urticaria in adults within my recollection at the present time are not of this type. These are often vigorous, energetic persons of nervous temperament who certainly cannot be accused of over-indulgence in food or drink. The error of diet from which they suffer is probably in the direction of taking too little food and at irregular intervals. The general instructions to be given to these patients are that their food must be fresh, well cooked, and easily digested; preserved articles of food are to be avoided with care. The starchy and sugary constituents of the diet should in all cases be regulated; it is not an infrequent consequence to find, especially in the case of children, that the limiting of the consumption of sweets, jam, etc., stops the urticarial outbreaks at once. After the ordinary diet has been investigated and if necessary properly arranged the use of occasional or accidental articles of diet should receive special attention. There are many instances in which one special article of diet used without consideration, or thought to be of little importance, produces the urticaria.

Internal medication is useful, and its principal purpose is to keep the alimentary tract free from fermenting and badly digested food and in as healthy a state as possible.

Local treatment is of the greatest importance, and the patient will expect you to make use of it immediately. The most effective of local methods of treatment is the use of baths. The baths should be given preferably at night before retiring to bed, though they may have to be used during the daytime also when there is much discomfort. An ordinary warm bath is sometimes sufficient to produce freedom from irritation, and the patient is able to go to sleep and rest. Usually, however, a better result can be obtained if the bath is medicated, and there are three methods of medication, all of which are useful.

(1) The bran bath, prepared by thoroughly infusing two, three, or four pounds of bran in a basin with hot water, pouring this infusion into the bath, making up the bath to a comfortable temperature, a little over body heat, and allowing the patient to stop in this for five or ten minutes.

(2) Alkaline baths, prepared by the addition of an alkali, such as potassium or sodium carbonate in the strength of 2 to 4 oz. to thirty gallons of warm water.

(3) Baths containing definite anti-pruritic substances, of which some of the forms of tar are most commonly used. Of these, perhaps one of the most satisfactory is the oleum betulæ albæ. This may be added and thoroughly mixed in an ordinary bath, or perhaps more easily in a bran

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bath. The amount to use in each bath will vary according to the effects, but it ranges from about 2 dr. to 2 oz. The liquor picis carbonis of the Pharmacopæia may be similarly used and in somewhat larger quantity.

Medicated baths of this character are often difficult to arrange in private practice, especially in the case of bran baths, when the disposal of the large amount of bran has to be arranged for. There are now on the market several preparations known as "bath powders," by means of which medicated baths can be arranged with much less trouble. Of these, the bath powder which is known as the "emollient bath powder" is a very good substitute for the bran bath. The powders are medicated by various drugs including varieties of tar, and these can be used with little trouble even in a private house where the bath appointments may not be all that could be desired. After the bath, and at various times during the day, local applications are necessary to restrain the itching and to make the patient comfortable. Of these local applications lotions are the most useful, and of lotions I may draw your attention to two classes especially:

(1) Alkaline lotions containing carbonate or oxide of zinc in suspension. The calamine lotion of our own Pharmacopæia may be considered as typical of this:

℞ Calaminæ præparatæ	. .	gr. xv
Zinci oxidi	. . . .	gr. x
Liquoris calcis	. . . .	♏lxxx
Glycerini	. . . .	♏xxx
Aquam	. . . . ad.	℥j

You will observe that it contains no oil. There is a preparation known as the linimentum calaminæ which does contain oil, and which is frequently confused with the lotion of calamine. You will find, however, as a rule that the non-oily preparation is much more satisfactory as a soothing application in cases of urticaria.

(2) The other lotions which are of special value are those containing lead. The preparations mostly in use are the glycerine of the subacetate of lead, and the simple solution of the subacetate of lead. These should be used freely diluted, and either in the form of an evaporating lotion or as a moist compress on the itching surface. There is a temptation to add to such lotions local anti-pruritic remedies, such as the preparation of tar, phenol, etc. Of these, carbolic acid is perhaps one of the most serviceable, but you will find that when it is advisable to use carbolic acid as an anti-pruritic, it is best to use it alone well diluted with water, or in the form of a dilute glycerine of carbolic acid. The anti-pruritic effect of carbolic acid will be obtained even when diluted so much as 1 in 100. It should never be used in strong solutions on account of its desquamative and irritating properties.

September 10th, 1906.

A LECTURE

ON

CHRONIC GASTRITIS.

Delivered at the Medical Graduates' College and Polyclinic.

By ROBERT HUTCHISON, M.D., F.R.C.P.,

Assistant Physician to the London Hospital, and to the Hospital for Sick Children Great Ormond Street.

LADIES AND GENTLEMEN,—I wish at the outset to make three statements about chronic gastritis. The first is that it is a rare condition. Some of you, I am sure, will be surprised to hear such a statement, but I am convinced of its truth. "Chronic gastric catarrh" is diagnosed far oftener than it should be. The ordinary case of habitual dyspepsia, believe me, is not a chronic gastric catarrh, as it is so often termed, but a neurosis. The second statement I would make about chronic gastritis is that it cannot be diagnosed with certainty from symptoms alone. It is one of the few conditions in which the use of the stomach-tube is almost imperative in order to arrive at an exact diagnosis. And the third statement I have to make about it is that it may run its course without producing any gastric symptoms at all. In other words, chronic gastritis may be entirely latent.

Etiology of Gastritis.

Now, in order that we may understand the nature of chronic gastritis and be able to prevent its occurrence, and treat it when present, we must first study the causes which give rise to it. There is, indeed, no disease of the stomach in which a diagnosis of the particular cause at work in producing the disorder is more important than in this. One can, I think, best understand the development of chronic gastritis by describing the mode of origin of a typical case of *acute* gastric catarrh. We may take as an illustration an actual case. A boy—for one sees gastritis best in young subjects, in whom it occurs more as a pure process, uncomplicated by changes in secretion—a boy goes to see a football match, and is there exposed to a cold wind whilst standing on damp ground. For lunch he has, as boys sometimes do, a hot mutton

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pie. He goes home and complains of gastric discomfort, becomes violently sick, has a high temperature and some rigors, soon develops acute diarrhœa, and in a day or two is well again. You have here a typical instance of acute gastritis, and there are brought out clearly in its production the two great causes of gastritis, whether it be acute or whether it be chronic, namely cold or chill on the one hand and irritation of the lining membrane of the stomach on the other. Now, everyone recognises irritation of the mucous membrane as a cause of gastric catarrh, but the part played by chill in its causation is much more apt to be left out of sight. I believe some people suffer from chronic gastritis in cold weather just as others suffer from chronic bronchitis, and that in this damp, cold, and changeable English climate the mucous membranes tend in winter to put on a slimy coat which in one man may take the form of a chronic bronchitis and in another of a chronic gastritis. In treatment, and still more in prevention, the factor of chill has always to be reckoned with.

The other factor, irritation of the mucous coat, may be exerted either mechanically or chemically. Mechanical irritation by unsuitable food is a common form. The swallowing of food which has been imperfectly chewed, for instance, sets up irritation, and hence chronic gastric catarrh may be maintained by a defective condition of the teeth. The mucous membrane may also be irritated by the habitual use of coarse, ill-cooked food containing hard particles. In some people the use of whole-meal bread, or of badly cooked vegetables is the cause of the irritation. In other cases the irritation is chemical, and here, again, its source may not be in the food itself but in the mouth. Many people with caries of the teeth and an unhealthy condition of the gums, with their mouths in a chronic state of sepsis, are swallowing bacterial products of an irritating nature, and many such suffer from chronic gastritis. Of chemical irritants in the diet alcohol is one of the most familiar as an exciter of gastritis, and there is no form of the disease which is better defined than alcoholic gastric catarrh. Other chemical irritants are condiments—*e.g.* mustard, pepper, and curry—and such articles as pickles and strong coffee. More potent even than these, perhaps, are the fatty acids often present in cooked fat, which has undergone partial chemical decomposition, with the liberation of butyric and other fatty acids. In the instance of the boy of whom I spoke, who lunched off a hot mutton pie, no doubt the decomposition of the hot mutton fat had much to do with the irritation of the stomach, which culminated in an attack of gastritis. In the matter of gastric irritation we have to remember that the personal factor or idiosyncrasy manifests itself strongly. Thus one man may be unable to eat a fragment of plaice, yet he can eat sole or turbot, or any other kind of fish, without any harm. There are often people, again, in whom eggs, or even a small portion of an egg, will excite gastric catarrh. We cannot say why, but stomachs do respond in this way to specific irritants. Passing now from the irritation caused by food, we must remember that chronic gastritis may arise from causes inside the stomach itself. You will readily understand how chronic gastritis tends to occur sooner or later as a complication in most forms of chronic dyspepsia. Suppose that a patient has for a long time an over-production of acid in his stomach; the chronic irritation of the mucous membrane so produced will ultimately lead, if it be allowed to go on, to gastritis. Similarly in cases of obstructive dilatation of the stomach, the retention of the contents, and their fermentation, with the production of organic acids and other irritating products, finally leads to such a condition of irritation that it passes on to a catarrh. Hence you may have to seek the cause of gastritis, not in the patient's mouth, or in what he eats or drinks, but in the vitiated condition of his own secretions.

Again, gastric catarrh is very apt to supervene when a patient's general nutrition is sufficiently impaired, and it comes on sooner or later in almost all cases of cachexia. You know that in malignant disease of the stomach free hydrochloric acid disappears from the gastric contents, and it was at one time supposed that this was due to a gastritis set up by the growth of the carcinoma. But it is now known that if you examine the stomach contents in a case of malignant disease in any organ, provided only that it has led to cachexia, you will find the free hydrochloric acid gone. In other words, malignant disease anywhere, if it has led to cachexia, produces a chronic gastritis. The same is true of the cachexia which is associated with profound anæmia, of which pernicious anæmia may be taken as the best example.

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Finally, you have to remember that chronic gastric catarrh is often associated with several conditions which lead to chronic congestion of the stomach-wall.

The chief example of these is found in chronic valvular disease of the heart with venous stasis. Such a patient has a congested liver and also congestion of the wall of his stomach, and one of the most troublesome features in such a case is often, not the heart condition itself, but the inability to get the patient properly nourished and to keep down medicines necessary to improve the state of his heart, owing to the vomiting which the chronic congestion of the stomach is so apt to cause.

Cirrhosis of the liver is also almost invariably associated with chronic gastritis, owing to portal stasis in the stomach-wall. Chronic nephritis is another condition which—not in this case owing to gastric congestion, but apparently from the escape into the stomach out of the blood of certain waste products which should be eliminated by the kidney—is a cause of chronic gastritis, and many cases of so-called uræmic vomiting have as their basis chronic catarrh of the stomach.

Symptoms of Chronic Gastritis.

Passing now from the causes of chronic gastric catarrh, I go on next to speak of the symptoms by which you can recognise it. You will remember that I said at the outset of this lecture that chronic gastric catarrh is one of those conditions which you cannot always recognise with certainty from symptoms alone. I said also that chronic gastric catarrh may run its whole course without producing any symptoms referable to the stomach at all. That statement may, perhaps, have surprised some of you, and I will therefore justify it by the description of an actual case. A patient, a labouring man æt. 50 years, came once to hospital complaining of chronic diarrhœa, which had been of some months' duration. He was a man of exemplary habits and character, and he could assign no cause for the occurrence of this diarrhœa, which had greatly reduced his strength. In answer to leading questions, he denied absolutely that he had had any symptoms of gastric discomfort at all. He said he took his food and digested it well without any inconvenience or difficulty. I gave him a selection of the usual astringents with a view to checking the diarrhœa, but with almost complete lack of success. After I had persevered in this way for some weeks I took him into the hospital for further investigation, in order to discover whether the intestinal condition might be secondary, as it sometimes is, to a perverted secretion of gastric juice. I gave him a test meal, and on drawing it off discovered that he was producing almost no gastric juice at all. The remains of the meal came up practically undigested, and the stomach contained in addition a considerable quantity of mucus. I then put him upon full doses of hydrochloric acid, and immediately his diarrhœa ceased. I have seen him at intervals since. He has an occasional recurrence of his symptoms, but immediately he takes the hydrochloric acid they stop. I have had several other cases of this disorder, which one may term "gastric diarrhœa," and which have been due to gastric catarrh of a chronic character, and yet in most, or at all events in a large proportion, of them the patient has denied having any symptoms of indigestion. The fact would seem to be that so long as the stomach preserves its motor power unimpaired it does not very much matter how much its secretion fails. Excess of secretion makes its presence known by pain, but diminished secretion is a silent disorder, and causes the patient no sensations by which he can recognise that anything is wrong. It is true that it is exceptional for there to be no stomach symptoms at all in chronic gastric catarrh, for in the majority of cases the patient will tell you that he suffers to some extent, but the symptoms are never very pronounced, and pain is conspicuous by its absence. If a patient complains of severe pain after eating, he is not the subject of gastric catarrh alone. Vomiting also is almost never present in these cases, though there may be retching and nausea, the former being often due, as in the alcoholic form of gastritis, not to irritation of the stomach, but to an irritable condition of the pharynx. Usually the patient will complain of defective appetite, and sometimes he will tell you he feels as if there were a weight in the stomach after taking meals, as if the food were not being digested quickly. Wasting and loss of strength may also be met with in gastric catarrh, but are not primarily due to the stomach disorder itself, but usually to the fact that the patient is taking an insufficient diet.

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Varieties of Chronic Gastritis.

From the symptoms which I have related you would not expect to diagnose gastric catarrh easily. They are largely negative, they amount only to a certain amount of discomfort and inconvenience, and hence one has to fall back upon the use of the stomach-tube to confirm the diagnosis. If you give the patient a test meal and draw off a sample, you will find in the majority of cases that you get evidence of diminution in the gastric secretion,—that is to say, you get a low degree of acidity. If the acidity be merely lowered, you may speak of the condition as *sub-acid gastritis*. Not long ago, for instance, I saw a patient, a member of our own profession, who had suffered from oppression and discomfort after food since his return from South Africa, where he served in the war. There was no vomiting or pain, and his appetite was fair, but in spite of that there was a moderate amount of wasting. On giving a test meal and passing the stomach-tube one drew off a comparatively small quantity of viscid contents containing an excess of mucus. The total acidity was 0·18 per cent., and there was no free hydrochloric acid present. On the other hand, the residue contained a large quantity of undigested food-particles. That may be regarded as a typical example of the sub-acid form of gastritis. Now, if the process goes on long enough, not only does the hydrochloric acid fall below normal, but it may disappear altogether. To those cases you may apply the term *anacid gastritis*. The case I described of the patient who suffered from gastric diarrhœa was an example of this form. In both those cases you will find, in addition to diminution of acid, the presence of an excess of mucus in the stomach. The contents are viscid and ropy, and on washing out the stomach in the morning you will find mucus present in it. It is these two things, the diminution of acidity and the presence of mucus, which are the marks of the great majority of cases of chronic gastritis.

There is, however, a third form of chronic gastritis, which does not correspond to this description, and that is the form termed *acid gastritis*. In acid gastritis you will find that in place of the acidity being diminished it is increased. You may have a total acidity of 0·3 per cent. or even 0·4 per cent. and an abundance of free hydrochloric acid. Here also you find an excess of mucus, so that there may be a very viscid, ropy condition of the contents after a test meal. The acid gastric catarrh cases are either those in which there is still so much active irritation of the mucous membrane that the glands are producing an excess of acid as well as of mucus, and the process has not gone on long enough to lead to their atrophy, or in which the gastritis has been grafted on the top of functional hypersecretion. To take again another actual case, which is a typical example of what I mean, that of a young man who, as a result of eating for some time an excess of highly stimulating food, especially curries, has brought about a condition of irritation in his stomach which has led to the over-production of acid, or, in other words, to hyperchlorhydria. He goes out insufficiently clad, and is exposed to a north-east wind. As a result of chill he gets a catarrh of the stomach. His appetite, hitherto excellent, fails, he suffers from nausea, and on examining the stomach contents there is now found an excess of ropy mucus in the stomach as well as an excess of acid. He now exhibits, in fact, a typical example of acid gastritis.

Diagnosis of Chronic Gastritis.

You may now ask me, Is the result of examining the stomach contents absolutely conclusive? Can you, after having examined the product of a test meal, say with certainty that you are dealing with chronic gastric catarrh? Well, there is only one thing which you may mistake it for, and that is diminished acidity from a purely nervous cause, a nervous hypochlorhydria or diminished secretion of gastric juice due to a neurosis—in other words, a functional arrest of secretion. Such functional arrest of secretion may take place acutely at any moment. I have known a man, on the receipt of sudden bad news in the middle of his dinner, lose all appetite and have to retire and vomit up all that he had eaten, which was found to be unchanged. This is an extreme example of nervous arrest of secretion. Again, in certain conditions of nervous exhaustion you may get a diminution of gastric secretion just as you may get enfeeblement of muscular tone, and such cases are not always easy to differentiate from chronic gastric catarrh. The presence of mucus may help you. In cases of nervous arrest of secretion, too, it is the acid that suffers and not the ferments, and if you test the gastric juice for the presence of rennet, as you can do by seeing

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whether it coagulates milk, you will find that in the nervous cases the ferment has not disappeared, whereas in gastritis it has. You may ask, again, how you are to recognise the supervention of gastric catarrh upon a case of dyspepsia from other cause. You will find in such a case that there is a disappearance or diminution of the acuter symptoms as soon as the catarrh has supervened. In a case of acid gastritis, for instance, the patient loses his pain when catarrh sets in, because the mucus which is poured out tends partly to neutralise the excess of acid. The patient's appetite also will almost certainly fall off. There may, too, be a very slight rise of temperature, causing a feeling of chilliness, and a very slight tenderness on pressure all over the stomach. Further, you will find that in such a case the tongue becomes furred, and there is a bad taste in the mouth, whereas those who have a pure neurosis do not usually have those signs; and lastly, you can confirm the diagnosis of the supervention of catarrh by giving a test meal. I would point out here that recognition of the inflammatory element in a case of dyspepsia which has primarily been due to other causes is of the greatest importance in treatment, because you cannot get the stomach right until you have got rid of the catarrh. You have first of all to subdue the inflammatory condition and then subsequently to deal with the error of secretion or motility.

Treatment of Chronic Gastritis.

And that leads me to speak of the treatment of a case of chronic gastritis. I have described its causes and symptomatology, and how you are to tell it from a neurosis, and how you are to recognise the supervention of a gastritis on the top of a previously existing ordinary dyspepsia, and now I have to tell you that the treatment of gastritis is not always an easy matter. The disease has a tendency to be exceedingly obstinate. As is the case with most of the mucous membranes of the body, once severe damage has been done, or any organic change occurred in the epithelial cells, it is very difficult to restore the mucous membrane again to its original condition. That, of course, is notoriously the case in bronchitis. A patient who has once had a severe bronchitis is very subject to suffer from it again on slight provocation. The surface cells of the epithelium in the bronchial tubes become damaged, and degrees of irritation which would be harmless in a perfectly sound individual become in such a patient sufficient to excite another acute attack of catarrh. It is so also with the stomach. Once a person has had one or two attacks of subacute or acute gastritis, he is very apt, on slight provocation, such as after a trivial error in diet, or a slight chill, to become again the subject of gastritis. Hence many of these people are patients with habitually weak stomachs, they are essentially chronic dyspeptics. The most you can hope to do for many of them is to give them such advice and guidance as will enable them to ward off any aggravation of their catarrh, and so prevent the process becoming worse than it is. Granted, then, that one has to deal with such a case, the first question which arises is as to the general treatment of the patient, Is he to be treated in bed or allowed to go about? Is he to have a change of climate? In considering these questions one has to be guided largely by the patient's general condition. If the patient is much reduced in health by the catarrh it will be advisable to send him to bed. My own opinion is that as a general rule dyspeptics are not sent to bed often enough. Rest in bed facilitates the treatment in every way. It enables the patient to get on comfortably with far less food than if he were going about doing his ordinary work. Moreover, being in bed keeps him at a uniform temperature and enables you to make the local applications to the stomach with greater convenience. The fact that cold or chills plays such a large part in the production of gastritis is another indication for putting the patient to bed. In the milder forms of course it is not necessary, and the patient may refuse to lie up, and in that case one must treat him as he goes about. Next we have to settle the question of diet. Here one may lay it down that the diet suitable for chronic gastric catarrh is one which shall be mechanically and chemically unirritating. The most bland food which can be given is milk, and in very bad cases it may be advisable to put the patient on that alone. All cases are benefited at the outset by it, even if they are only given it for a week, because there is no food which causes the stomach less labour. In the less severe cases, on the other hand, one would give milk mixed with farinaceous foods and so on; through every degree of severity one can build up a diet suitable to the particular case, always remembering

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that it must contain nothing stimulating, such as spices and condiments, and that it must contain no mechanically irritating particles which are not easily brought into solution. The latter condition is best fulfilled by passing the vegetable foods through a sieve and mincing all animal foods.

With regard to the question of local applications to the stomach, I think we do not make sufficient use in these cases of moist heat. Poulticing has gone out of fashion even in the treatment of bronchitis—perhaps it has gone too much out of fashion—but I am certain it is not used nearly enough in dyspepsia. If the patient be in bed one can apply continuous poultices for a week, but in milder cases great good can be done by applying a hot fomentation over the stomach during the night, whilst an abdominal bandage is worn by day. The wearing of an abdominal flannel belt should be insisted upon in all cases where the patient is going about.

The next question is as to change of climate. I think there is no doubt that many patients with chronic gastritis are as much benefited by change of climate as patients with chronic bronchitis are. At all events, in an obstinate case one should try the effect of sending the patient to a warm, dry place, where there are no cold winds. This is specially advisable in the spring months.

Lastly, I want to say a few words about drugs. I am afraid drugs are not of much assistance in treating chronic gastritis, but if they are properly chosen they will do something to bring about a cure. Mercury you will often find of considerable help. In using it the point to attend to is to avoid giving large doses. About one tenth of a grain of calomel—which sounds almost homœopathic—or one fifth of a grain, given every night or every other night, for a time, followed by a draught containing sulphate of soda in the morning is often of service, and it is usually advisable when dealing with a case of ordinary dyspepsia in which gastritis has supervened to begin the treatment by such a course. Another useful prescription is two grains of blue pill and three or four of compound rhubarb pill every night.

Seeing that gastric catarrh tends to be accompanied by failure of secretion and diminished appetite, the patient will be helped by bitters and small doses of alkalies before meals. Rhubarb is also of great benefit: I do not pretend to know how it is, but there is no form of chronic dyspepsia in which one gets better results from its administration than in chronic gastritis, and it may be given in combination with other drugs which I have already mentioned. If there be much irritability of the stomach or nausea, one should add bismuth to the prescription. Rhubarb and bismuth with soda and a bitter make a very suitable combination given before meals as a stimulant to appetite, and as a solvent of mucus, and probably also, from the action of the rhubarb, as a mild astringent. In some cases, again, one gets the best results from giving hydrochloric acid after meals. In the case of the patient whom I described as suffering from chronic diarrhœa the benefit derived from hydrochloric acid was immediately apparent. Sometimes one can add a ferment to the hydrochloric acid with advantage. I told you that in the advanced stage of chronic gastritis the production of ferments tends to be limited just as the production of acid does, and in such cases the administration of an active peptonising ferment is indicated. There are very few conditions of the stomach in which ferments are of use, and the administration of pepsin is probably over-done, but the cases in which it does good are those in which the production of ferments has undergone diminution. The difficulty is to get an active preparation of pepsin. There are a few on the market. The preparation known as ingluvin has certainly given me good results in some cases. I have prescibed it in the cases of doctors who were themselves the subjects of gastric catarrh, and who were convinced that it made a great difference when added to hydrochloric acid, and as these are trained observers their evidence is of value.

You have therefore two plans open to you when prescribing for a case of chronic gastritis—either to give alkalies and bitters (with or without the addition of rhubarb and bismuth) *before* meals in order to stimulate secretion, or else to give hydrochloric acid and a ferment *after* meals in order to supplement the natural gastric juice. Which of these is the best plan to adopt in any given case I do not pretend to be able to tell you. I think you must usually regard the matter as a therapeutic experiment and try first one plan and if that fails fall back upon the other.

September 10th, 1906.

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A POST-GRADUATE LECTURE

ON

PROLAPSE AND PROCIDENTIA.

Delivered at the Ancoats Hospital.

By E. STANMORE BISHOP, F.R.C.S.,
Hon. Surgeon Ancoats Hospital; Surgeon for Diseases of Women, Jewish Hospital, Manchester.

GENTLEMEN,—Allow me once more to welcome you to Ancoats Hospital on this the occasion of the first of our second series of Post-graduate Demonstrations.

It is one of the advantages of a hospital that from time to time we may take stock, so to speak, of any particular condition, collect together and compare cases of any one kind, and if it is one the treatment of which has undergone any changes, realise more completely than would otherwise be possible the advantages or disadvantages of the various methods employed, and determine what progress has been made and what improvements, if any, are possible in the future.

Such stock-taking cannot fail to be of advantage to the operator himself as it confirms and gives a reliable basis to his opinions, which previously may have been merely a general impression, whilst should the condition be a common one, one constantly met with in ordinary every-day practice, the results arrived at and the reasons for the conclusions formed should, I think, also greatly interest and be of importance to those who first meet and are called upon in the first instance to advise upon them.

These Post-graduate Demonstrations are admirably adapted for such a comparison of results and for their communication between us; and such a condition is uterine prolapse, and its extreme condition, procidentia, and therefore I have chosen it for consideration at this our first meeting.

I wonder if it has ever struck you to ask, not why any particular woman should be the subject of prolapse of her uterus, but why all women do not sooner or later suffer in this way. The condition is common enough, as we all know; but still, all women do not suffer, not even all parturient women, not even women with large families, not even all women who have undergone instrumental labours. On the other hand, some unmarried girls suffer. I do not here refer to the well-known elongated cervix seen in some girls, but to true prolapse of a normally sized uterus.

In September of last year I was asked by my friend Dr. Riddall of Longsight to see with him a young lady, æt. 18 years, who had startled all her female relations by a protrusion between the lips of the vulva, which Dr. Riddall quickly diagnosed as being the uterus. The history given was that two months previously the patient had felt sudden sharp pain in the right inguinal region, whilst sitting in the house: after a short time this disappeared, leaving a sore sensation, which lasted a few hours.

Three weeks before examination she had noticed a protrusion between the labia when walking. As she was an enthusiastic tennis player it was at first thought that this exertion might have brought about the displacement.

Four days before being seen by Dr. Riddall this returned. There was no pain, but the prolapse was increased by any exertion, and did not again disappear.

Attempts at reduction were followed by acute retention, requiring the catheter. On examination under chloroform an ovarian cyst was found wedged in the upper outlet of the pelvis and pressing the uterus downwards. This was removed, and the patient recovered, with perfect return to the normal state.

If you consult the text-books you will find that the uterus is said to be retained in position by its ligaments—the sacro-uterine, the broad and the round ligaments—by the firmness of the folds of Douglas, by its attachment to the bladder, by the valvular character of the vagina and the action upon that canal of the levator ani, by the intact condition of the pelvic fascia, the two layers of which enclose that muscle, and by the transversales perinei and the "body" of the perineum, lastly, by some authors, by the "retentive power" of the abdomen, whatever that may mean. Now, all these were in their normal condition in this case and yet prolapse occurred. Moreover the patient was young, and therefore all her tissues were resilient and in no way deficient in tone, as her speedy recovery proved. The prolapse was in this case due unquestionably to the increased and increasing pressure of the impacted cyst above; but the case shows, I think, that, given sufficient

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intra-abdominal pressure above, none of the structures mentioned are in themselves able to prevent prolapse of the uterus.

Clearly, then, one of the first things we have to eliminate in any case where prolapse is present is anything which may increase that intra-abdominal pressure beyond the normal. Loaded intestine, producing constant straining to get rid of hard fæces which have been allowed to accumulate in the colonic sacculi, and which eventuates in the production of small rocks which have to be evacuated by great effort, is one very important item. Compression of the abdomen by tight unyielding corsets, great accumulation of fat in and about the intestines, enteroptosis, alcoholism, which acts by increasing the volume of the liver—these and numerous other causes will naturally occur to you.

But when all has been done in the way of purgatives and enemata to unload the colon, exercise to keep down fat, and improve the muscular tone, removal of tight corsets, and the use of a rational method of dress, etc., there will still remain a certain amount of intra-abdominal pressure which will act upon any weak place and tend to produce protrusion of the organs contained in the abdomen.

So that the question resolves itself into this: Which of the many factors mentioned as preventing prolapse have most effect in counteracting this intra-abdominal pressure, which is always present? and by what operation can we best avail ourselves of them or reproduce them?

Some years ago a great deal of stress was laid upon the position of the uterus in these cases. It was said that all cases of prolapse began with a retroposition of the fundus uteri, that this was the first step in such cases. The body of the uterus, which normally lay at an angle with the cavity beneath, was thus placed more directly in the axis of the latter, and therefore was driven as a wedge into it farther and farther downwards. There can be no doubt that retroposition, and especially retroflexion, especially if associated with adhesions fixing the uterus in this position, produces many unpleasant sensations to the patient, and that theoretically the process of prolapse should be easier when the uterus and vagina are in the same line, but I cannot help thinking that this condition has been unduly emphasised and for these reasons:

First, the uterus is normally a movable body, capable of normally lying in a retroposition.

Second, many cases exist in which well-marked retroposition can be demonstrated, where no tendency to prolapse has ever shown itself, even when observation has been extended over years.

Third, most procident uteri show no traces of retroflexion, which condition should be most effective.

Fourth, after reduction and operation by colporrhaphy, most uteri lie in retroposition although no tendency to recurrence takes place. Schultze says "the position of the uterus after colpoperineorrhaphy or any other successful operation for prolapse is generally retroversion or retroflexion," and he explains the absence of any symptoms calling for further interference in order to remedy this condition, which before the operation had been credited with so much importance, by saying that the woman has suffered so long from major miseries that relief from them overshadows all minor troubles, but he advises that a patient should wear a sledge pessary in order to counteract this tendency.

I say I think that in relation to prolapse too much stress has been laid upon retroposition of the uterus. I do not say that in itself it does not produce suffering or call for interference.

But the influence of this idea was so strong, that in or about 1890 and the decade that followed, many of us, following Alexander, were shortening the round ligaments which pull the fundus uteri forwards, very frequently with the idea that in this way the tendency to prolapse would be prevented.

Of course it is evident that such a proceeding can have no effect in the way of raising a uterus already prolapsed, since, even if the ligaments were shortened to their fullest extent, they could not possibly raise the fundus higher than the pubic spines to which they were attached, and could, therefore, produce little or no difference in the height of the body of the uterus. Moreover, the other attachments of the uterus, especially the sacro-uterine ligaments, being also relaxed or destroyed, the effect was frequently to pull the whole uterus forward to the pubic arch, where it hung still more directly in the line of the dilated vagina below.

The next step was to secure the uterus itself in such a way that it should not be possible for it to descend, and accordingly it was hung up to the abdominal wall as the most convenient fixed point.

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Under the names of "ventro-fixation" and "ventro-suspension" numerous operations were and are still performed with this object, some cases of which I am able to show, though the usual difficulty with hospital patients of change of residence prevents my showing many, for my last case was done in 1900. [The patients were shown and the position of the uterus demonstrated.]

I think you will agree with me that the result is not ideal, even for old women who are past the child-bearing period, whilst for younger women, who may again be pregnant, the result is full of peril. I know that certain operators persistently minimise these risks; they are, however, real enough. During pregnancy the whole uterus should develop and dilate. That portion of the uterine tissue which is fixed develops, but does not dilate; a large cushion of the muscular wall is thus formed, which impedes the rotation and descent of the child in parturition, whilst the rest of the uterus has to dilate to a greater extent than the normal in order to make up for that segment which is now useless for that purpose. The excessive dilatation of the free portion may pass beyond the limits of safety, and cases are on record in which rupture of the over-thinned muscular wall has occurred.

The uterus is normally a movable organ, and any operation which has for its end, as all plastic surgical operations should have for the end at which they aim, a *restitutio ad integrum*, a return to the *status quo ante*, should leave the uterus still a mobile organ. Ventrofixation can never do this.

It was found also that in some instances where the anterior abdominal wall was thin and flaccid the result was not so much to hold the uterus up as to drag the abdominal wall inwards and downwards.

It was curious, however, that a result which might have been expected, decrease in the capacity of the bladder, was seldom or never produced.

When casting about for some measure which should avoid these manifest disadvantages, and considering the many normal attachments of the uterus, my attention was naturally directed to those which act upon its posterior and inferior surface, the sacro-uterine ligaments. These would naturally act at a far more favourable angle, inasmuch as by pulling up the cervix backwards the body and fundus of the uterus must rotate upon the broad ligaments as upon an axis in the desired direction forwards. In addition, as they would act upon the lower part of the body we desired to lift, the upper part would be forced to precede it in an upward direction.

All writers agree that relaxation of the folds of Douglas is one of the earliest, as it is one of the most important, of the factors in the production of prolapse; but I had been taught that these folds, sacro-uterine ligaments as they were called, were simply folds of peritoneum. Now, all my surgical experience of the peritoneum went to show that that membrane was one of the most extensible of all membranes present in the body, that whilst it was rebellious to sudden force, preferring rather to split than to give, slow persistent or constantly recurring gentle pressure found it excessively amenable. Any one who has experienced in himself or has carefully watched the growth of a scrotal hernia knows that no effective resistance is to be expected from it, but that when once the protrusion has passed beyond the resistance afforded by the fibrous tissues of the inguinal canal, there is scarcely any limit to the distension, gradual and painless, which it may not undergo. If, then, one had merely to rely upon the resistance of such peritoneal folds, the case was hopeless, since, shorten them as we might, they would only stretch once more.

But Dixon and Cunningham, in the 'Journal of Anatomy and Physiology,' 1902, showed, as indeed had Schultze before them, though his description had not attracted the attention deserved, that these ligaments were composed of true muscular and tendinous tissue, lying below and between the peritoneal folds which covered them, so that in the normal person there were true ligaments acting in the direction desired, and therefore that an attempt to reproduce them might have the desired effect.

I did not attempt to dissect out these delicate muscles and reunite them as Bovee in America claims to have succeeded in doing. Most of these cases have lasted for years before we see them, and such fine muscles, once ruptured, atrophy, retract, and disappear. The search for them is predestined to fail; but knowing now that such fibrous attachment had once existed, and had been effective, I strove

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to imitate them and to construct a new sacro-uterine ligament from the vaginal wall which would be sufficiently tough and fibrous a structure to bear the superposed weight. By uniting the posterior fornix, previously deprived of a portion of its peritoneal covering, to the sacrum behind, at a point where injurious pressure upon the rectum might be avoided, I desired to bring about that firm union which Greig Smith showed was produced when peritoneum and fibrous tissue were brought together. The area of vagina lying between this line of union and its normal attachment to the uterus would permit of sufficient mobility of the cervix whilst yet firmly supporting that organ at the normal height; moreover it would hold the lower pole of the uterus backward and effect the forward tilting of the fundus which was desired.

On the cadaver the practicability and apparent effectiveness of this proceeding was easily demonstrated. By means of a special sound the posterior fornix was made to protrude behind the uterus in Douglas's pouch, and was supported, when, through a median abdominal incision, the parts could be reached without difficulty, the patient being in the Trendelenburg position. I then carried out the method on ten patients, and the result was so good that in 1903 I demonstrated the operation before the British Gynæcological Society. Some of the patients were lost sight of, and for anything I know are still in good health and relieved from their troubles. One or two, however, came back to me with a relapse. The point of union had yielded once more and the prolapse returned. Probably the area of union was not broad enough. There was no mortality. I might perhaps have remedied this faulty union by improved technique had not my attention been arrested by another operation, originally devised, I believe, by Dr. Donald, on the lines of Hegar's method of colporrhaphy, which has so far given me such good results that for the present at least I have abandoned my own operation in favour of his.

It is very often the case that comparison of an abnormal condition of one part of the body with a similar condition in another will throw new light upon the first or on both. It will be seen that the two problems to be solved have been approached from different sides by different minds, that, similar as the conditions are, certain points have attracted certain operators effectively in the one instance which have been ignored, or at least have not been accorded the same importance by those who have attempted to remedy the other.

Such a similar condition to that of prolapsus uteri is the one met with in inguinal hernia. It will be readily recognised that the uterine condition is really a hernia of that organ, that in both there is the same valvular canal to be passed through by an organ which is free to move above it, but is incapable of entering that canal as long as its valvular character is preserved. Has it equally been recognised that a loop of small intestine approaches the internal ring at almost exactly the same angle that the uterus bears to the vaginal canal, and that the loop is prevented from entering that passage mainly by the action of the abdominal muscles which preserve the potential character of the inguinal canal and prevent it from becoming an actual one? So long as the inguinal passage is closed by the firm pressure of the muscles around, and is only potentially open, no bowel escapes, though the internal ring is there to admit it; but when once the bowel has effected an entrance, the constantly recurring impulse produced by coughing or straining slowly but irresistibly opens out the canal below and results in the production of a hernia.

May not the production of a prolapsed uterus be explained in much the same way?—the essential factors of such production, I mean. There are many differences which will readily occur to all of us, but these, I suggest, are of minor importance, and only modify but do not contradict the analogy I am endeavouring to make clear.

If you look at a vertical frozen section of the female pelvis which has not been altered by parturition, it is evident that the vaginal canal is valvular and potential, that there is no actual cavity below the os uteri. The walls are elastic and resilient, the posterior is firmly pressed against the anterior, and is maintained in this position partly by its own tonicity, but also greatly by the action of the levator ani, which draws upon its two outer borders so as to flatten out the ring which the walls themselves would naturally form and transform it into a horizontal slit, just as traction upon the sides of an elastic ring would do. For this traction to be effective the muscular fibres themselves must

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be at a certain tension. If you enlarge the ring the fibres lose that tension and cannot act with the same force, the walls remain ring-like, the posterior wall no longer is forced to fit firmly against the anterior, and the passage from being merely potential becomes actual.

The vagina is normally sufficiently small to allow of these muscular fibres acting upon its sides with their full force. During parturition they are stretched by the passage of the child. If the labour is normal they are not unduly stretched or torn, and spring back again with more or less celerity to their normal size when the levator ani fibres can again act with their normal tension. Should the labour be a prolonged one, or too soon repeated before the normal calibre has again been reached, they may never return entirely, but remain relaxed, and therefore patulous, since the levator can no longer act effectively, its insertion and origin being thereby approximated. It is evident that as long as the vagina remains enlarged in size its anterior and posterior walls can never be in contact, and therefore the way is open for intra-abdominal pressure to drive anything from above into it. Whether that shall be the uterus or intestinal loops in Douglas's pouch or bladder in front will depend upon other factors which need not now be considered. The way is open below for the descent of some viscera of some kind.

If this is so it is evident that the course of events is this:

First, enlargement of the vaginal canal.

Second, consequent relaxation and ineffectiveness of the levator ani.

Third, consequent loss of the valvular character of the lower passage; and the process of surgical restitution to the normal state must follow the same lines.

First, narrowing of the vaginal canal, so as to allow the levator ani fibres inserted into its sides to regain their normal tension, and by pulling upon those sides to restore the original valvular character of the canal.

Incidentally it will be noted that in considering the prevention of such a patulous condition the obstetrician should take into consideration, when conducting a labour, the question whether prolonged contact with the forecoming head may not so stretch the vagina for a long period that it cannot recover, and, therefore, whether the earlier interference by instrumental aid may not be indicated with a view to prevent after-troubles of this kind.

But to return to the surgical treatment of these cases. What are the methods at present available and in every-day use?

Naturally our first thought will be of pessaries. From the earliest times attempts have been made to shore up the descending uterus, and many pessaries are devised having for their *raison d'être* simply that idea. Such appliances are balls, rings, and the old pot pessary. If the ideas previously sketched are correct, it is abundantly evident that these can only perpetuate and intensify the mischief since the vagina becomes only the more stretched by them if they are to fulfil their purpose. The only pessary which is, I think, admissible is some form of the Hodge pessary, which acts by leverage, and the best method of applying that is this copper wire covered by celluloid which can be bent into any form which the ingenuity of the surgeon may consider best suited to any individual case.

But no pessary can restore the normal state; all pessaries are but makeshifts and surgeons can never be content with them.

What operations are available and how far do they restore the normal condition?

To judge between them it is well to formulate what is expected of them.

First, and most important of all, they should restore the vaginal canal to its original size and shape, so that the pull upon the sides of that canal by the levator fibres can reproduce the flattened valvular character which is so important.

Second, they should reunite the sphincter ani if that has been divided.

Third, they should reunite the transverse perinei and the ends of the constrictor vulvæ, so as to form a solid, firm perineal body.

Fourth, they should correct the retroposition of the uterus.

I take it for granted that you are all acquainted with the technique of the various operations I shall name. It would take far too long to go into their details.

Kelly says that "the normal supports of the normal uterus and vaginal outlet can never be perfectly restored," and this mental attitude would appear to be a common one in writers upon this subject. It will account for many of the proceedings proposed, including Kelly's own. He advises a resection of the relaxed vaginal *outlet*, restoring its calibre, changing the direction of its axis and changing its position, associated with a supra-vaginal amputation of the cervix; in bad cases the abdomen is opened and the uterus suspended to the anterior abdominal wall. Here you have the key-note of most of the rest. Decrease the size of the *outlet*, change its axis, and if that does not keep up the uterus lessen its size and weight, and if that

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fails hang up the uterus itself. Any one can see that though such narrowing of the outlet may prevent the external appearance of the uterus, it can in no way prevent its descent as described, and all the more important symptoms which accompany such descent, such as are produced by dragging upon the sympathetic system of nerves, the bladder, and the peritoneal attachments above.

Tait's flap-splitting operation has practically the same end in view, and produces similar results.

Many operators appear to deeply respect what is called the posterior column of the vagina and to look upon it as being really what its name implies, a pillar or strong support of the structures above. Thus Bischoff's operation, described by Schültz, leaves this untouched, removing the mucous membrane from the sides of the vagina and leaving the central column intact. So does that of Graily Hewitt. Kelly's operation is very similar. Neither carry their incisions beyond what has been called the crest of the vaulted prominence of the rectocele in the centre, but upwards on each side for about an inch and a quarter above the outlet. Emmet's operation is the basis of both. Winckel's operation is simply the formation of a bridge or shelf across the lower part of the vagina. It is plain that the idea in all is to narrow the *outlet*, and from the description of Emmet's operation in Hirst's 'Gynæcology' it is also evident that it was believed that the fibres of the levator ani were themselves torn through and that by this operation they were again united. I want you to look at this drawing, which gives a view of the levator not often seen, I imagine, and you will readily understand that these fibres, being inserted on the outside of the canal, cannot be torn, unless the vagina were split at the sides by the original cause, and probably not even then, but that the increased calibre of the canal permits their relaxation, the approximation of their line of insertion into the canal to the white pelvic line from which they originate, and therefore throws them out of action. No fibres, or only a few scattered ones, pass actually between the vagina and rectum. The main effective ones act only by their tension on the sides of this passage.

Of course from a cursory outside view of the perineum these operations appear to produce an admirable æsthetic effect. Even the usual test of many surgeons, the thickness of the perineum, of whatever composed, is satisfied; but the true test of success in the *reductio ab integrum*, the true preventive to relapse, the reproduction of the valve-like vagina, is not attained. Moreover none of them alter in any way the malposition of the uterus.

Hegar's operation is far more effective, though still imperfect. He removes a triangle of the posterior wall from the cervix downwards, and this is the basis of the operation which appears to give the most perfect results. I have not been able to find any printed description of Dr. Donald's method, which that operator very courteously allowed me to see him perform. It consists of two superposed triangular denudations of the posterior wall, the apex of the upper one lying immediately below the cervix and the truncated apex of the lower being a continuation of the base of the upper with a wider angle. In addition, should any cystocele be present, a similar but single reversed triangle is removed from the anterior wall. If retroversion is present, as it so generally is, an anterior colpotomy is done, and the peritoneal flap covering the fundus and anterior wall of the uterus is drawn down and fixed to the vaginal wall in front. This brings forward the uterus and turns it into a position of anteversion; the raw surface of the bladder produced by the colpotomy becomes adherent to the anterior peritoneal surface of the uterus, and maintains it in that position though the uterus still remains mobile. When the colporrhaphy is completed as far as the base of the upper triangle, the lower is closed by deeper sutures which re-unite any fibres of the levator which pass behind the vagina, the two perinei, and the constrictor of the vulva. In this way not only is a sound perineum built up, but the original valvular character of the vagina is restored throughout its whole length and the fibres of the levator which act upon its lateral surface restored to their original tension. It is, I think, the most complete, scientific, and effective operation at present in use, and it fulfils admirably all the indications formulated at the commencement. I have very great pleasure in being able to show you the results which I have been able to obtain by its use. If you pass a speculum you will see how the canal closes firmly in its whole length on the withdrawal of the instrument. Such a canal is indeed the best possible preventive of any relapse because it is the nearest possible approach to the original state.

I think that the only method to which I have not referred in this paper is that of Edebohl's, which he described at the recent meeting of the British Medical Association in this city. I only mention it now to condemn it. This operator in certain cases where all else fails—in his hands—removes the entire genital tract and builds up a solid perineum; in fact, he transforms the woman into a castrated man with a perineal urethral fistula. Such an operation may truly be described as a method of despair.

I trust, gentlemen, that this short review of the operative treatment of uterine prolapse, embodying as it does the results of some twenty years of operative work, and the reasons which have influenced the adoption of this, the latest method, and the rejection of the rest, has not been without interest to you and may serve as a groundwork for your own researches on this subject.

September 10th, 1906.

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LECTURES

ON

THE TREATMENT OF SURGICAL EMERGENCIES.

Delivered at the West London Hospital Post-Graduate College.

By ASLETT BALDWIN, F.R.C.S.Eng.,

Senior Assistant Surgeon, Lecturer on Practical Surgery and Teacher of Operative Surgery at the West London Hospital.

LECTURE I.

GENTLEMEN,— I will with your permission commence with smaller matters first and gradually work up to more serious ones, as the former are of great importance and interest in our every-day work.

Foreign bodies in the ear.—The first subject I will bring to your notice is that of foreign bodies in the ear, which are mostly met with in children, if we except the plugs of wax and wool found in the ears of adults. One must first of all ascertain for certain that a foreign body is present before adopting any means other than syringing to remove it. Much trouble has been caused by neglect of this precaution. It should be seen through a speculum. It may be possible in a child who is sensible and not too nervous or frightened to get rid of the body by syringing with warm water or boric lotion. This should in all cases be tried first and will succeed in most cases. When syringing the ear the auricle should be drawn upwards and backwards in order to straighten out the passage. It should be noticed if there is anywhere a space between the body and the wall of the meatus; if so, the stream of water should be directed along that wall and through the opening so that the fluid may get behind the body and push it out. The section of the passage is oval, so there may be a space above and below the body. In any case, the

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stream of water should be directed along one or other wall of the meatus. Sometimes changes in the patient's position may assist expulsion. For instance, the patient may lie on a table with the head projecting over the end and the ear pointing downwards; the surgeon will then, of course, have to syringe upwards. If this is not successful or where a child is frightened or perhaps has been hurt and bleeding from previous attempts at extraction, it is best to give a general anæsthetic and then see exactly in a good light the condition of affairs. If blood or discharge is present it must be carefully syringed away with warm water, then with a ten-volume solution of hydrogen peroxide; this very effectually removes blood and discharge and also acts as a hæmostatic. Adrenalin may also be used for the latter purpose. If the body still resists removal with the syringe, the part should then be got as dry as possible with little pledgets of absorbent wool held in forceps. Now, suppose the body to be a glass bead with a hole through it, there is great difficulty in seizing a body of this sort with one of the forceps made for the purpose; they will probably only push it further in and should not be used. The best instrument is one, such as a dentist's excavator, with a fine point bent at right angles to the shaft of the instrument and the right-angled portion must be short, or a cystotome or similar small home-made hook fashioned of wire. The bead should be rotated with the point of the instrument till the hole comes into view, if it cannot be seen already. The right-angled portion is then passed through the hole, and the bead is extracted by traction without difficulty. It may be possible to pass a bent probe or a loop of wire past a foreign body and then to draw it out, or alteration in its position may make this possible. For this purpose pressure with the finger "upon the skin close to and in front of the tragus, carried upward and around the meatus, upon the auricle, and back again to the starting-point, when the manœuvre is repeated several times," has been recommended. It is also credited with having caused extrusion of a foreign body.

If the body is a smooth one, with no projection and no hole in it, and it cannot be caught or syringed out, its exposed surface must be completely freed from grease; if this has not been done by hydrogen peroxide it can be done by gently rubbing with a little piece of wool soaked in ether. A clean camel-hair brush which has had its point dipped in a solution of shellac in ether is passed to the body and made to envelop it as much as possible; wool is then placed round the ear to hold the brush in position and a bandage placed round the head. The brush will adhere firmly to the bead and will withdraw it when removed in several hours' time. Seccotine or glue or similar adhesive material will also answer the purpose. A small sharp hook is sometimes useful in removing a vegetable body such as a pea, the hook being made to catch in the body before withdrawing it. Seeds may swell after being some time in the ear, and their extraction may be assisted by a preliminary soaking in alcohol, which absorbs their moisture and causes them to shrink. Insects may be removed by syringing with warm water. Larvæ may have to be killed before using the syringe or it may fail to dislodge them. This may be done by filling the ear with warm alcohol, or placing a few drops of chloroform on a small piece of lint in a wineglass and pressing the open mouth of the latter on the side of the head, enveloping the ear so that the vapour gets into the meatus. This is a very useful method also for the relief of pain in the ear.

If, when the case is first seen, the auditory canal is inflamed by injudicious attempts at removal of the body it will be wisest to wait several days for the inflammation to subside, provided there are no urgent symptoms present, the ear being gently syringed with a weak warm antiseptic lotion several times daily and then carefully dried with absorbent wool. It is important to remember that there is usually no urgency about these cases whatever, and that foreign bodies have often remained in the ear for long periods of time without doing any harm; in fact, the greatest danger associated with them attends attempts at their removal by unskilled persons. From time to time cases have occurred where the surgeon has deemed it necessary to separate the auricle behind, and chisel away part of the bony meatus for the removal of an impacted foreign body, but they are few and far between. Experienced surgeons have imagined that a body was in the tympanum when it was not so; I saw this happen a short time ago. A foreign body in the tympanum has been removed by washing it out with a stream of water directed through a Eustachian catheter. If this

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failed to remove the body it would probably alter its position so that it could be removed by one of the means already mentioned. It is also well in these cases to bear in mind that a general anæsthetic should always be given to a child if anything more than an examination or the use of the syringe is to be attempted, and even for these one will be necessary in many cases.

Foreign bodies in the nose.—I have usually removed foreign bodies from the nose with a small blunt, slightly bent Volkman's spoon, or a strabismus hook, without anæsthesia. If the body is far back a general anæsthetic may be needed, and it will then be advisable to put the left index finger behind the posterior nares while removing the body, to prevent its falling back into the larynx. In cases of unilateral nasal discharge, especially in children, it should be remembered that it may be due to a foreign body and search made for one with a probe; it may be necessary to use local or general anæsthesia to do this successfully, as the body may be hidden by granulations and the cavity of the nose extremely tender; reflected light from a forehead mirror and a nasal speculum will be required. It may be possible to remove a foreign body from one nostril by directing a stream of water under low pressure down the other nostril, the patient meanwhile breathing vigorously through the open mouth, but the majority of children will not allow of this and the bent spoon or strabismus hook will generally be successful. Syringing should not be attempted if there is any discharge present as it may cause infection of other cavities, but larvæ may be removed in this way after being killed by a ten per cent. alcoholic spray.

Foreign bodies in the œsophagus.—Coins in the œsophagus can be removed without an anæsthetic. The child should be wrapped in a rug or something of the kind to confine its arms and legs, and laid on its back on a table, with its head hanging a little backwards over the edge to make the mouth and œsophagus as much as possible in the same line. An assistant stands on the child's left side and holds its mouth open with a gag. The surgeon with his left index finger holds the child's tongue down and passes the coin-catcher along the back wall of the pharynx and down past the coin. When caught it is drawn steadily and gently upwards; it often sticks a little at the tip of the œsophagus, when traction a little to one side and then the other will disengage it. The coin-catcher which I much prefer has the part which catches the coin fastened directly to the whalebone handle without the intervention of a flexible spring or a piece of metal, and should be hinged at its connection, so that whichever side of the coin it passes it will catch it. This kind of instrument is passed much easier than the others and is not so likely to break. It is very important to test the instrument before using it to see if the catcher part is firmly attached to the handle, or it may come off and be lost in the gullet. Not so very long ago the catcher part of one of these instruments, together with the flexible piece of metal which attached it to the handle, broke off while being used in this hospital, and had to be removed from the stomach by gastrotomy. In some cases the X rays give help in the removal of coins. The shadow of the coin or other body opaque to the rays is observed by means of the fluoroscope, and instruments can be guided to the foreign body with great precision, the manipulations being guided by the shadows. The fluoroscope consists of a screen, which becomes luminous when the X rays fall upon it, enclosed in an instrument much like an old-fashioned stereoscope. Its advantage is that a dark room is not required, so that the administration of an anæsthetic, if one is necessary, is rendered much safer and the work generally is much facilitated.

Small tooth plates without hooks may perhaps be extracted through the mouth with forceps or a coin-catcher, or passed into the stomach, but large or impacted plates must be removed by œsophagotomy if it is possible to reach them that way, as attempts to drag them out would lead to laceration of the œsophagus and fatal cellulitis. The operation is usually done through an incision in front of the left sterno-mastoid muscle. The œsophagus should be opened along the whole length of the plate, care being taken not to injure the recurrent laryngeal nerve, which lies in the groove between the œsophagus and the trachea, and any hooks or projections which have become caught in the mucous membrane disentangled or cut off. Extraction may be made easier by cutting the plate into two or more pieces. No attempt should be made to keep the plate whole as a "specimen" if by so doing injury is caused to the œsophageal wall.

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When the plate is removed the wound in the œsophagus should be completely closed with iodised catgut * and the rest of the wound partly closed and drained, after being well flushed out with 1 in 2000 perchloride of mercury lotion. If possible, the patient should be fed by the rectum for a week or more. The teeth and mouth should be washed with a non-poisonous antiseptic solution several times daily and a small quantity should be swallowed each time. Such a solution is chinosol, 1 in 2000, peroxide of hydrogen, 10-vol. solution, or acetozone, 10 grains to a pint of water. This is done to bathe the wound internally and to keep it as aseptic as possible.

Foreign bodies in the larynx.—Foreign bodies in the larynx should be removed if possible by endolaryngeal methods, aided by cocaine locally and suitable forceps, hooks, or loops of wire. The site and character of the body should be ascertained first by examination with the laryngoscope and the attempts at removal assisted by the same instrument and a good light. If the dyspnœa is urgent tracheotomy should be done at once. In some cases of extreme urgency it may be necessary to do laryngotomy or tracheotomy by one thrust of a pen-knife or whatever knife is available, the airway being held open by rotating the knife, or holding the wound open by retractors made of bent hairpins, the bent portion being at the looped end of the pin, or whatever object is available for the purpose.

When the foreign body cannot be removed from above, tracheotomy should be performed and means taken to prevent blood passing by the side of the tracheotomy tube into the trachea. This may be done by packing gauze round the tube, or by using such a tube as Hahn's or Trendelenburg's or Semon's. The cricoid and part or, if necessary, the whole of the thyroid cartilages can then be split in the middle line and the body gently pushed up into the mouth. It has been recommended when the thyroid has to be split completely to make a transverse groove across the cartilage before its division to insure absolutely correct approximation of the two halves afterwards, or the voice will be rendered permanently hoarse. The tracheotomy tube should be left in for several days because of the danger of œdema of the glottis.

If an adult has attacks of suffocation and a foreign body in the larynx is suspected but nothing can be found and no disease can be seen in the larynx to account for it, and there is no interference with the recurrent laryngeal nerves caused by such things as carcinoma of the œsophagus, aneurysm of the aorta, tuberculous disease of the lung, tumours in the chest, etc., the patella and ocular reflexes should be examined, as the trouble may be a symptom of tabes dorsalis—in fact, a laryngeal crisis. Or there may be abductor paralysis of the vocal cords and secondary adductor muscular contracture, when a slight catarrhal inflammation of the mucous membrane may lead to further narrowing of an already greatly narrowed glottis and any little extra muscular effort is liable to bring on an attack; the patient might be quite unaware of the paralytic condition of his cords, and the condition comes on most insidiously and meanwhile gives rise to no symptoms. A patient afflicted in this manner should be kept quiet and free from muscular exertion and mental worry; he should breathe air as pure as possible and free from dust, so that the larynx is not irritated in any way. The attacks are treated and prevented by spraying or painting the interior of the larynx with a 10 per cent. solution of cocaine, or as much as 20 per cent. may be used. Probably other local anæsthetics, as stovain, or eucain and adrenalin, would also be efficacious. In some cases death has taken place during these suffocative attacks, in others life has been saved by a prompt tracheotomy. In bad cases the question of tracheotomy will always have to be considered, especially when the patient cannot count on skilled assistance being at hand for the application of cocaine to the interior of the larynx. One application will generally ward off an attack for about twenty four-hours. It should be remembered that paralysis of the internal thyro-arytenoid muscles may come on later in such a case; if this happens the chink of the glottis will be fusiform and the danger of suffocation disappears, but no one can say if the paralysis will come on or not, and meanwhile the patient may have a fatal seizure.

Foreign bodies in the trachea.—If a foreign body

* This is prepared by soaking the catgut for ten days in a solution of iodine in iodide of potassium, one per cent. of each; it can be stored dry in a well corked or stoppered bottle.

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is in the trachea the best plan is to do a high tracheotomy and then, holding the trachea open by sutures or two sharp hooks, to see if the body will be coughed out; this may be aided by tickling the inside of the trachea with a sterilised feather. The opening into the trachea must be a very free one so that the body can escape easily. If it is not driven out it may perhaps be caught by a suitable instrument. You must be on the alert to see that it is not sucked back again if it presents itself at the opening. If not successful the patient should be inverted and shaken, the tracheal wound being held widely open meantime. If the body cannot be got rid of, the tracheal wound should be kept widely open by Golding Bird's trachea dilator for a time, in the hope that it will escape.

Foreign bodies in the bronchus.—In attempting the removal of a foreign body from the bronchus, a free opening should be made in the trachea as low down as possible. The edges of the tracheal wound should be held widely open by strong wire sutures and an opportunity given to the body to be coughed out. If you get a case of this kind to deal with, you must be very much on the alert to catch the body as soon as it appears, or the next inspiration after the expiratory effort which caused the body to appear on view may drag it back again out of sight. Coughing may be encouraged by tickling the inside of the trachea. Of course it is very important that anything introduced into the trachea should be absolutely sterile, or septic broncho-pneumonia may be set up. If the body is not coughed out, the patient should, if possible, be inverted and shaken. If these means are not successful an attempt may be made to seize the body with Durham's forceps, which are flexible and can be bent as required, or stout wire may be bent to an obtuse angle, about an inch or so from its end, and the end bent up into a small hook; the other end of the wire should be bent into a ring, which must be in the same plane as the bent portion of the wire and the hook, in order to know the whereabouts of the latter when out of sight. If the trachea is large enough the surgeon's finger may be of use in guiding forceps or a blunt hook down to the body. It will most likely be in the right bronchus, but this will have been ascertained beforehand by physical examination of the chest, and if possible by the X rays. If the foreign body were opaque to the X rays they and the fluoroscope might be useful in guiding an instrument down to it. If attempts at removal are not successful, they should not be persisted in too long, but the trachea should be kept open and the body may be got rid of. After a considerable interval it may be coughed out, weeks or months afterwards. Golding Bird's trachea dilator is a useful instrument for this purpose and would give the body a much better chance to escape than if a tube were in the trachea. If the body is impacted so low down that it cannot be reached from the tracheotomy opening the patient's chances are but slight, although a successful case has been reported where a boot button was removed from the left bronchus, after incision of the chest wall and removal of parts of several ribs, also another where an abscess formed in the lung, which became adherent to the chest wall; after incision into the abscess the body escaped.

Hæmorrhage after tooth-extraction.—I will now speak of several kinds of hæmorrhage which may cause trouble. In hæmorrhage from a tooth socket, first of all blood and clots should be completely removed so that you can see exactly from whence the blood is coming; it may be coming from deep down in the alveolus or from the edge of the gum round the socket. I saw a case of this kind a short time back. The patient was a publican who had been bleeding all night; his mouth was full of dark brown blood-clot; he was pale and frightened and presented a pitiable plight. On clearing out his mouth it was easily seen that the hæmorrhage came from the edge of the torn gum and not from the socket at all. Pressure on the bleeding point with the finger for a minute or two completely stopped the bleeding. If the hæmorrhage is from a tooth socket it should be completely cleared of clot and then tightly packed from the bottom with a strip of gauze which should be folded as a pad over the socket so that when the teeth are closed it will be held in position and the pressure maintained. A handkerchief should be tied round the head and lower jaw to assist this. If the opposite tooth is absent or the pressure unsatisfactory a piece of cork cut in the right shape tied to a string and placed in position will get over the difficulty when the jaw is closed on it. Before inserting the end of the gauze into the cavity it may be dipped into a styptic solution, such as adrenalin, peroxide of hydrogen, or turpentine.

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Hæmorrhage after tonsillotomy.—When bleeding persists after excision of tonsils in spite of sucking ice, the mouth should be cleared of blood-clot and the back of the mouth sprayed with cocaine, 10 per cent. solution. A good number of forceps should be ready holding little dabs of wool and in a good light the bleeding tonsil rapidly dried and the bleeding point seen. A dab which has been dipped in a mixture of equal parts of tannic and gallic acids should be then pressed well on to the tonsil, counter pressure being made on the outside of the neck with the other hand. Any other suitable styptic may of course be used. If this is not successful and it is available and possible to use it the actual cautery at a dull red heat may be tried; or by means of a curved needle held in a holder a suture may be passed through the anterior and posterior pillars of the fauces so that when tied the bleeding will be stopped by its pressure.

Hæmorrhage after removal of adenoids.—Hæmorrhage after removal of adenoids can be stopped by forcing into the naso-pharynx a clean sponge or dab of wool which has been soaked in peroxide of hydrogen 20-volume solution or adrenalin and chloretone 1 in 1000 solution, or tincture of hammamelis or haseline. It need not have a string attached to it and can be easily removed in about twelve hours' time by one of the forceps used for the removal of post-nasal adenoids.

Epistaxis. — Epistaxis frequently takes place from the anterior part of the septum of the nose, and may be controlled by pressure on the outside of the nose with the finger, the patient sitting bolt upright. Pressure on the upper lip just below the nostril will control the small branch which goes from the superior coronary artery in the upper lip to the septum. If this pressure has to be kept up for more than a short time it is easily done by placing a pad on the lip and tying a broad piece of tape round the face and head supporting the pad in position. Cold to the nose, cold to the back of the neck, elevation of the arms above the head may cause contraction of the vessels and arrest of the bleeding. The latter should not be tried for more than two or three minutes or the vessels will dilate and the hæmorrhage be increased. Letting the patient sit with the feet in hot water is useful, care being taken that he is not allowed to faint. If the bleeding point can be seen it may be touched with the galvano-cautery at a dull red heat, after the local application of cocaine, which itself tends to arrest the hæmorrhage; or chromic acid, or nitrate of silver fused on the end of a probe may be used in the same way. When measures such as these fail a long narrow strip of sterile muslin or similar material soaked in a 15-volume solution of peroxide of hydrogen or adrenalin and chloretone solution 1 in 1000, may be packed into the nose in successive layers from behind forwards with a blunt probe, or small wool swabs tied to pieces of silk may be packed into the nostril in a similar way. These packings should not be allowed to remain longer than twenty-four hours. By far the most convenient and safest instrument I know of for plugging the nose is Rose's nose tampon. This consists of a central stem like a short English gum-elastic catheter, a little longer than the distance between the anterior and posterior nares; surrounding this is an indiarubber bag, which can be blown up by a small pump when the instrument has been introduced into the nose. The air is prevented from escaping by turning a small tap, after which the pump is removed. After the instrument has been in place for a few hours, some of the air can be let out, to see if the hæmorrhage has ceased; if so the instrument is withdrawn, if not, it can be blown up again at once. It can be withdrawn after deflation, cleansed and reinserted in a few seconds and with a minimum of discomfort to the patient. Septic troubles are much less likely to follow its use than after any other method of plugging with which I am acquainted. Some time ago I believe I saved a man's life by its use. He was somewhat addicted to alcoholic liquors, and a week before I saw him had been ejected from a public-house, his nose coming into violent contact with the curbstone outside. His friends said he had been bleeding the whole week. He was extremely blanched and was nearly dead, the hæmorrhage still going on. The tampon was inserted and at once arrested the hæmorrhage. In a few hours' time some air was let out, but as hæmorrhage took place, the instrument was blown up again. In about twelve hours' time it was removed, cleaned, and replaced as the hæmorrhage had not ceased. The next day, or day but one, the instrument was again partially deflated, and as no hæmorrhage took place it was emptied and withdrawn, after which there was no further loss of blood and the man slowly recovered.

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There are several other tampons for the same purpose, but this one is quite satisfactory and is cheaper than the others.

Hæmorrhages from scalp wounds.— Bleeding arteries in a wound in the scalp, if they cannot be controlled by the sutures, should be seized with forceps and tied before sewing up the wound. If this is impossible owing to retraction of the vessels, they should be underrun by a suture in a fully curved needle passed through the whole thickness of the scalp down to below the level of the vessels, after the scalp has been shaved and cleaned. When the sutures are tied the hæmorrhage is arrested. The underrunning stitch can be removed in a few days' time with the other sutures. A few years ago it was thought that wounds of the scalp should not be completely closed with sutures owing to the danger of sepsis. Now after thorough cleansing and disinfection with mercury lotion the majority of these wounds are sutured up entirely with perfect safety.

Hæmorrhage after excision of tongue.—If the ligature slips after excision of the tongue, the person in charge of the case should at once pass the left index and perhaps the middle finger beyond the stump of the tongue, and draw it firmly forwards; this will control the bleeding. The vessel can then be picked up with an artery forceps and ligatured.

September 17th, 1906.

BLEPHARITIS.

A Lecture delivered at the Polyclinic.

By N. BISHOP HARMAN, M.A., M.B.Cantab., F.R.C.S.Eng.,

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GENTLEMEN,—This trouble seems a very small one wherewith to occupy an hour's talk. But it is one of those little things that matter a good deal, particularly in these days of keen competition, when the personal appearance of a man may, and certainly does in woman, turn the scale of fortune. We cannot afford to neglect any genuine appeal for aid in any personal deformity, and there is many a fair face just spoiled by a little pink rim to the eyelids; and to those who know, that pink rim is the outward and visible sign of a defective action of the eyes, and consequently a diminished capacity for daily work.

To understand the disease we must consider the structures affected by it. The eyelids are just folds of skin possessing all that equipment, epidermis, hair, and glands, which we recognise as possessed by the skin everywhere; but in the eyelids both the skin and its equipment are modified to play their special part. On their outer and exposed sides the skin is thin, soft, and very supple; it is very loosely attached to its supporting tissue, so that when that is drawn up, as in elevation of the upper lid, the skin is thrown into horizontal folds which fit beneath the brow better than any patent blind within its frame. On the inner or ocular side the lid is stiffened with a plate of tough connective tissue, and lined with an epithelium evenly besmeared with mucus, the secretion of its own cells, so that the lid glides over the equally smoothly covered globe with a nicety that could not be excelled.

But there is a zone betwixt these two inner and outer surfaces—the edges of the lids, those parts of the lids, upper and lower, which make brief but very frequent contact day in and day out for a lifetime, and without—in healthy people—the least sign of stress.

It is worth while trying to appreciate how nice must be the adjustment and equipment of the lids to realise so excellent a result as this. In the course of a single waking day of, say, fifteen hours the lids of an eye come in contact with each other—blink—something like 5000 to 20,000 times. I am inclined to think that if we tapped the edges of our hands together as often as this they would show signs of soreness; but the eyelids do not, and this immunity is the result of the arrangement and specialisation of the sebaceous glands.

There are no scattered sweat-glands or sebaceous glands on the conjunctiva, they are not needed; the conjunctiva is unctuous by reason of its inherent mucous secretion. The sweat-glands have become the lachrymal glands and are tucked away in the orbit, whence they issue their bland secretion to wash the eye. The sebaceous glands are ranged in rank with their mouths to the edges of the lids, where alone there is danger of friction. If you turn over an eyelid you will notice shining through the pinkish conjunctiva a row of short, yellowish

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lines vertical to the lid edge; and on the thickness of the edge itself, with or without the aid of a lens, you will see a row of small holes ranged parallel to the cilia on their inner rank. These yellowish lines are the sebaceous glands, which we know under the name of Meibomian glands, and the small holes we notice ranged along the lid margins are the mouths of the glands.

To the secretion of these glands is due the freedom of the lid margins from irritation as the result of the frequent blinking. The secretion coats the margins with a layer of smooth grease, the margins do not come into contact cells with cells; the grease is not sticky, so the lids open as readily as they close; and lastly, and by no means least, the grease coats the margins with a natural waterproofing, so that so long as the secretion is healthy the margins are not wetted by the tears.

When this secretion is defective in these qualities there follow those symptoms and changes in the lids which we know as *blepharitis* or *ciliary blepharitis*.

Sociology.

Blepharitis is one of the commonest of eye diseases amongst children. In five months of 1903 I examined the eyes of 22,000 children in London elementary schools. I found 351 cases of eye disease, and of these 270, or nearly two thirds, were varieties of blepharitis. The disease is more common amongst children than amongst adults. I examined the case papers of the patients attending one clinic at Moorfields in 1902, and found the ratio of cases of blepharitis was as follows: Infancy to 3 years, 300; school children 4 to 14 years, 259; young adults 15 to 30 years, 45; adults 31 to 55 years, 10; elders over 56 years, 2. The disease is more common amongst very young children than in older children. This is well demonstrated by the age incidence of cases seen at the Belgrave Hospital during ten years; at the age of four years the incidence is at its maximum, the cases rapidly increase to this age and decline as rapidly after this age.

The disease is much more common amongst that part of the population who are habitually dirty or ill-kept than amongst the clean. In examining the schools I classified them as containing children who in the mass were of average cleanliness, or above or below this average of cleanliness, and I found that the percentage of eye disease worked out as follows: Above average cleanliness, 0·873; average, 1·416; below average, 1·92—that is, disease was twice as frequent amongst the dirty as the clean. Again, in examining the Moorfields cases, I separated alien from native patients, knowing from experience that the native patients were on the whole cleaner in person than the alien. I found that the ratio of incidence of blepharitis in the alien was 126·5, as compared with 15 in the native patients. It is, therefore, clear that the disease is fostered by dirt.

The disease is most common amongst those people who are possessed of skins and hair which are both thick and coarse. They are folk who either exhibit or are found to be liable to "blackheads," pustules, acne, and seborrhœa. In fact, blepharitis falls into the same group as do these diseases of the skin.

The disease in its chronic form is rarely found among those in whom the refraction of the eye is normal. It is almost always associated with an error of refraction.

Etiology.

One great factor in the causation of the disease, especially as it affects children, has been noted, namely, dirt. This probably is the principal explanation of the maximum incidence of the disease at the age of four years. It is just at this age that immature humanity begins to "find itself." It has its own idea of play, generally marked by grovelling in any dirt that can be found, and then rubbing as much of the dirt as may be into eyes, nose, and mouth; and particularly into the eyes, when, as in the nature of the case is certain, it gets hurt and cries, and helps the operation with both dirty fists well screwed into the eyes.

Moreover, it is about this age that many of the milder exanthemata blossom and the coincident catarrhs of conjunctiva and nasal mucosa not infrequently leave traces, of which one is blepharitis.

The lashes of the eyelids and the mouths of the big Meibomian glands are well adapted to harbour micro-organisms, and it will be patent to all how difficult, if not impossible, it is to clean these parts by ordinary washing. If, then, we notice dirty faces and bodies, how much more dirty these parts which so easily harbour dirt. I examined the flora

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of the eyelids of twenty children, ten chosen by their master as clean children and ten as dirty. I found in all cases numerous colonies of cocci, the *Staphylococcus albus* or *S. epidermidis albus* for the most part, but there were many colonies of the *aureus* and *subflavus* varieties, and in one case several colonies of the Morax-Axenfeld bacillus, which is a well-known cause of a definite form of conjunctivitis. There were, on the average, 10 per cent. more colonies of organisms in the dirty children than in the clean; also, the colour-producing cocci were most numerous in the dirty children, and these organisms are the most active factors in producing pustular inflammations such as blepharitis.

I have noted the frequency in which blepharitis is a sequela of exanthemata, particularly measles. It is in these cases almost always acute; it rarely becomes chronic unless there are other exciting causes. The same observation holds good for blepharitis produced by any precedent inflammations of skin or conjunctiva of any order.

The Course of the Disease.

The chronic form is that which mostly concerns us. It may be associated with an acute attack due to some conjunctival or skin inflammation; but by no means infrequently—indeed, probably in the majority of the cases I am about to describe—the commencement of the affection is so quiet and insidious that it is not noticed by the patient. It is only when the condition becomes so advanced as to be remarked by the sufferer or others that the desire of human nature to ascribe everything to a cause leads to the formulation of a definite "previous history," though the assigned "cause" may be *post hoc*, and by no means *propter hoc*.

In examining children I have found it possible to demonstrate every stage of the disease in a single large school. In the earliest stage there is only a little dry, white, bran-like material fixed on to the lashes, just like papers on a file. The Meibomian secretion is excessive, and is not carried away by cleansing with sufficient rapidity, so it dries and crusts around the lashes. If these cases be seen late in the day, after much reading and particularly after doing fine sewing, the margins of the lids will be pink instead of yellowish white, and the rate of blinking will be found to be increased.

Later the injection of the margins—the "weakness of the eyes"—is noticed all day long.

There is redness, swelling, and tenderness of the margins; they feel sore and prick, so they are frequently rubbed, and the blinking is excessive. Then the lashes become crusted and clogged together with dried secretion, *débris* of cells, and foreign matter. There is an alteration in the secretion of the Meibomian glands; it is excessive and thin, it loses in its waterproofing quality, so that it no longer efficiently protects the lid margins. The irritation is followed by an increased lachrymation, and the edges of the lids, being no longer properly protected by the Meibomian secretion, are always being wetted, become sodden, and the tears tend to overflow on to the skin of the lids.

As the crusts accumulate about the lashes there is formed an excellent breeding-place for the abounding cocci, small ulcers form under the crusts, and if we remove them we find raw bleeding surfaces. The disease spreads along the shafts of the lashes into the follicles, the lashes die, are shed and not replaced. There is set up a vicious circle; the blepharitis predisposes on even slight provocation to conjunctivitis and eczema of the skin, and these in turn exaggerate the blepharitis; ultimately both cilia and Meibomian glands may be destroyed.

The chronicity may be such as to induce a hyperplasia of the fibrous basis of the cutis vera, producing numerous sessile fleshy knobs along the lids. I show you a drawing of such a case found in a young Jew; the upper palpebral margin had a row of semi-pedunculated fibrous growths, which presented a disgusting appearance, the lower lid was everted and raw, the cilia were almost gone.

In most cases, however, the disease drags out a miserable course for years, until there is left a soft, smooth, pink pouting, rounded edge to the lid. The margin becomes completely atrophied as regards the proper constituents of the region; the skin passes into the conjunctiva by imperceptible gradations of epithelial thinning, and the cutis vera is replaced by a felt of soft scar-tissue.

Soil, Seed, and Crop.

In studying the sociology of this disease there are noted three outstanding features:

(1) The subjects of the affection are most frequently possessed of skin and hair which are

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both thick and greasy, and they either suffer from or are liable to "blackheads," pustule, acne, and seborrhœa. There is, then, a susceptible SOIL.

(2) The subjects of the affection will, on the objective examination of their refraction, most commonly be found to show an error, generally of an astigmatic variety. There is in this factor a PREPARATION OF THE SOIL.

(3) Lastly, there is the never-failing presence, in even the cleanest of persons, of pyogenic organisms about the lid margins, staphylococci of one or many varieties. There is, then, plenty of SEED ready to hand for sowing.

It is not difficult to link into a connected story the relations between this susceptible soil, the preparation of the soil, and the seed. The clue, to my mind, particularly in those long-standing intractable cases in older school children and young adult life, lies in the second factor, the preparation of the soil. The defect in vision may be very slight, but when the child is put to close and difficult work, especially when from unhealthy general conditions of life fatigue is readily induced, the tiring of the eyes, the suffusion of tears, cause repeated blinking, so that the margin of the lids tap, tap, tap against each other with far greater frequency than is normal. The soil, naturally susceptible, is slightly irritated by the mechanical stimulation and prepared for the reception of the seed, which probably makes its first implantation in the hair-follicles.

The seed will flourish in direct proportion to its plentifulness—that is, in proportion to the dirtiness of the child's surroundings and person. This is eloquently shown in the statistics gathered from the board schools and in the comparison of native and alien patients at Moorfields. With the alien there is also more frequently the susceptible soil—the thick, greasy skin and coarse hair of the Southern melanochroi, in contrast to the thinner skin and finer hair of the Northern xanthochroi.

In subjects better circumstanced there may be both the susceptible soil and the preparation of the soil, but their greater cleanliness so reduces the seed that the occurrence of blepharitis amongst them is rare, and, when found, of but slight degree.

TREATMENT.

To cure a disease dependent on three clearly defined causes, all of which are well within our control, should not be difficult, and it is not, provided the cure be undertaken in due time. The plan of the cure may be drawn up with delightful brevity and precision—make the patient clean and remove the cause of irritation.

The poor are dreadfully afraid of removing these crusts from the eyelids. "They bleed," they protest, and so they do when ulceration has begun. But every scrap of crust must come off.

The frequent bathing of the eyelids with an alkaline lotion—simple bicarbonate of soda—will soften and dissolve them; plenty of soap and water and friction with a flannel will do it just as well. But a good clean-up by the surgeon himself, and a final light swabbing with a weak solution of silver nitrate, will do more than gallons of lotion liberally bestowed during many weeks. Then the application of a smooth antiseptic ointment will keep the lids soft and reduce the liability to the collection of more crusts and scabs. Plain boric ointment in yellow vaseline, or a doubly diluted ointment of ammoniated mercury, or the yellow oxide of mercury in a mixture of lanoline and vaseline to the strength of 1 per cent., are all of value, the last perhaps most of all.

These means will do much to reduce the unsightly ravages made by the disease, but they will not touch the thing which in my experience is most frequently the real cause of the irritation. To get at this the refraction of the eye must be made out, objectively, by retinoscopy, and any irregularity found must be corrected with glasses.

I am no lover of spectacles. I do not think them an order of distinction of a superior humanity. I wear them myself, and should detest them did they not give me perfect vision. I am always sorry when I see children bespectacled. But it is a choice of evils—spectacles or worse. Certainly spectacles are decorations as compared with the raw, red, lashless eyelids of blepharitis. The wise surgeon will measure his patient, and as he finds the patient careful or careless so he will amend his instructions: the careless will have to wear spectacles constantly; the careful may in many cases escape with the injunction to wear them for all work requiring long-continued and fixed attention of the eyes—for reading, writing, sewing, and for concert, theatre, or church.

The soil—the thick, greasy skin and hair—cannot be amended, but the condition of it can be; regular baths, scrupulous cleanliness of hands, hair, and clothing will do much to reduce the susceptibility of the worst soil.

September 17th, 1906.

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THE SURGICAL TREATMENT OF HEPATIC CIRRHOSIS.

By CHARLES GREENE CUMSTON, M.D.

In a recent issue of the 'Birmingham Medical Review' I published a paper relative to the value and indications of omentopexy in cases of cirrhosis of the liver with ascites, but I now intend only referring to drainage of the biliary tract in certain cases of hepatic cirrhosis. The surgical treatment of this condition has been simple exploratory laparotomy, laparotomy combined with destruction of adhesions, with or without massage of the biliary tract, and lastly, laparotomy followed by cholecystostomy.

We will rapidly consider the most essential facts relative to the pathogenesis of hepatic cirrhosis, which may be defined as a generalised proliferation of interstitial tissue, accompanied by various pathologic changes in the hepatic cell. In other words, a cirrhosis merely represents a chronic hepatitis which is the ultimate stage of an irritative process of the liver, having for its starting-point the biliary tract or the blood-vessels. It is at the present time an established fact that infection is the primary etiological factor in certain cases of cirrhosis, the infectious agent being propagated from the intestine to the liver by way of the biliary tract. Hypertrophic biliary cirrhosis with chronic icterus represents the most perfect type of this morbid condition. But, in order to fully understand this question of hepatic pathology one should not forget that, roughly speaking, the liver may be reduced to an hepatic cell, united to the intestinal tract by an excretory canal. The latter is in direct continuity, both anatomically and physiologically, with the hepatic cell and represents one of the means of draining the liver, and this duct is in direct continuity with the intestine and partakes of the pathological vicissitudes of the latter, and it may be safely stated that the sclerous process is the result of microbic invasion from the gut.

In the normal condition the biliary tract may be considered as absolutely aseptic; and this normal asepsis also exists in the hepatic parenchyma, a very great contrast with the intensely septic condition of the intestinal mucosa, which abounds in micro-organisms. I would, however, add that the last portion of the choledochus is normally the abode of two organisms, the *Bacterium coli* and the *Staphylococcus aureus*. There consequently exists at the point of the anastomosis of the biliary tract in the intestine a transitional zone whose microbic flora is practically certain. As to the intestine, it is, on the contrary, the ordinary home for a large number of bacteria; and Gessner, as far back as 1889, made the statement that he was always able to discover the following organisms: (1) the *Bacterium tholocideum*, a short oval bacillus, coagulating milk, and pathogenic for mice; (2) the *Bacterium coli*; (3) two other bacilli, one of which liquefies gelatine; (4) two staphylococci, one yellow and the other an orange-yellow, liquefying gelatine; (5) the *Streptococcus pyogenes duodenalis*, in all probability analogous to the *Streptococcus pyogenes aureus* so well known in pathology.

From this fact the biliary tract is in constant danger of infection from the numerous intestinal bacteria; and since the digestive tract is open to all kinds of external infections, it may become infected occasionally by the most varied kind of bacteria. Consequently when the frontier which separates the aseptic canalicular zone from the infected intestinal zone is involved, an ascending biliary infection results. The bile and the walls of the biliary tract then offer all the necessary conditions for the culture and development of the various organisms, an angiocholitis being the first step of hepatic cirrhosis, the process being thus definitely created.

Before discussing the anatomical changes set up in the liver by chronic infection of the biliary tract I shall endeavour to show that the part played by infection in the pathogenesis of biliary cirrhosis is based on precise facts. If, in the first place, we examine the bacteriological researches which have been undertaken on the bile of patients afflicted with cirrhosis, it at once becomes evident that the examinations have been invariably positive. For example, in Gilbert's case an organism was found which differed from Escherich's bacillus in some of its characters, while in another instance an organism similar to the *Bacterium coli* was discovered, and in Michaux's case the bile was infected by the colon bacillus, likewise in the case recorded by Pauchet. The presence of numerous pneumococci was easily made evident in Lejars' case. In my own cases the *Bacterium coli* was present in two, and this organism in association with the *Staphylococcus*

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aureus was found in the third. Pus has even been met with in the liver of cases of hypertrophic biliary cirrhosis, and Sabourin, as far back as 1884, described a suppurating angiocholitis in this affection. A cloudy and purulent fluid has occasionally been found in the gall-bladder in cases of cirrhosis; and Hanot, Charcot, and Gombault consider that hypertrophic biliary cirrhosis is in close relationship to those instances of hepatic cirrhosis following biliary lithiasis or after experimental ligature of the choledochus when the biliary tract has been infected by bacteria. And still more, Charrin and Roger have been able to reproduce the lesions perfectly in their experimental work by directly injecting living or sterilised microbic cultures into the choledochus, while Gaston in 1895 developed the theory that Hanot's disease to a certain extent realises the perfect type of infectious liver. In his remarkable memoir on cirrhosis Chauffard, after having put aside intoxication as the probable cause of the sclerotic process, declares that many plausible facts plead the cause of infection *a priori*, and he adds that at a given time it surely intervenes in the form of an ascending biliary infection, even a pyogenic one, whose starting-point must certainly be the intestine. Gilbert and Surmont unhesitatingly unite in one group the biliary cirrhoses which are, according to their way of thinking, infectious, and they consider Hanot's disease as a consequence of an ascending infection of the biliary tract by bacteria coming from the intestine. From all this one can only conclude that the infection is foremost in the etiology of the sclerosis process. Then again, it may be added that in the affection that we are now discussing one will find the lymphatics of the hilum of the liver hypertrophied, as well as an hypertrophy of the spleen, leucocytosis accompanied by paroxysmal elevation of temperature, all of which are evident manifestations of an infectious process. The presence of bacteria having been demonstrated in biliary cirrhosis, two theories have been discussed based upon this fact. In the first a real pathogenic part is supposed to be played by the bacteria, which in themselves alone are sufficient to engender an infectious sclerosis, while the second hypothesis is that hypertrophic cirrhosis is accompanied by secondary infection. It matters very little, however, what explanation is admitted, and it is none the less true that at a certain time in the process there is infection.

Let us now consider the pathogenesis of the glandular and secretory apparatus of the liver, set up by the evolution of the infectious process. I have already pointed out that the biliary tract, which is aseptic in the normal state, is constantly menaced by the intestinal bacteria which are held in check by the physiological functions of the bile, and especially the hydraulic pressure resulting from the flowing off of this fluid. Now, if for any reason this equilibrium is interfered with, the intestinal germs will invade the biliary tract and the infection will undergo its evolution under the form of an ascending catarrhal and obliterating angiocholitis. This catarrhal process ends in sclerosis of the biliary apparatus.

At the International Medical Congress in 1897 Gilbert and Surmont expressed themselves as follows: "L'infection peut s'être limitée aux gros conduits, à la vésicule; la paroi biliare n'est pas toujours atteinte dans toute son épaisseur et avec la même intensité; la muqueuse peut avoir été seule lésée et même uniquement dans sa partie superficielle. On conçoit donc la possibillité d'un retour à l'état normal ou de la persistance d'altérations insignifiantes. . . . Que l'on envisage par contre une de ces infections atteignant les voies intra-hépatiques, persistant sous forme de poussées pendant des mois et des années; les lésions des canalicules, l'infiltration leucocytique des zones biliaires, l'apparition des cellules fusiformes à leur niveau, l'élargissement des espaces portes et la formation des néo-canalicules, l'envahissement conjonctif inter et intra-lobulaire: tels sont les résultats anatomiques successifs d'un tel processus; tels sont, en particulier, dans un certain nombre de cas, les résultats des angiocholites calculeuses. Elles aboutissent à la cirrhose biliare. Il faut ajouter que dans ces circonstances la cellule hépatique reste pendant longtemps intacte ou qu'elle subit même l'hypertrophie et l'hypergénès. Ainsi se trouvent donc réalisées des lésions identiques à celles qui caractérisent l'affection qu'Hanot a individualisée sous le nom de cirrhose hypertrophique avec ictère chronique. Dans les deux cas les processus pathogénique et le processus anatomique sont les mêmes; mais ici, il s'agit d'une cirrhose biliare infectieuse spontanée ou sans obstruction, et là d'une cirrhose biliare infectieuse avec obstruction."

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To sum up, Hanot's disease is merely a subacute, ascending, catarrhal, obliterating angiocholitis, susceptible of becoming combined with cirrhosis, the latter being only a secondary phenomenon added to the lesion of the biliary ducts. This is why in biliary cirrhosis the result of microbic invasion the particular form of the process after a time sets up irritative lesions rather than pyogenic ones of angiocholitis and chronic radicular peri-angiocholitis, and for this reason it is quite natural to foresee the excellent results that drainage of the infected biliary apparatus can give.

A word now as to venous cirrhosis, because one or two such cases have been submitted to surgical treatment, and in one at least the condition of the patient was very markedly improved after drainage of the biliary tract, the ascites did not recur, the icterus disappeared, and the general condition was greatly improved. This happy result causes me to question whether or not it is not logical to admit that infection also plays a part in this type of hepatic sclerosis where the agent of infection reaches the liver by way of the blood, and the part played by infection appears to me most plausible on account of the fortunate result obtained by operative interference. I would also recall in this respect that certain authorities have generalised the doctrine of infection to venous cirrhosis—thinking, for example, that *a priori* in cardiac cirrhosis, or the hard, nutmeg liver, it is very unlikely that one is dealing with disturbances in the circulation only, and that there must also be concomitant infection or intoxication, as has been expressed by the authors in the 'Traité de Médecine,' edited by Brouardel and Gilbert. These same authorities state farther on that it has been experimentally demonstrated that if biliary cirrhoses are of an infectious nature, vascular cirrhoses may be either toxic or infectious.

In considering the indications for operative treatment I will consider separately hypertrophic cirrhosis with icterus and hypertrophic cirrhosis without icterus; the first corresponds to the affection known under the name of "Hanot's disease," while the second represents a class of badly defined chronic hepatitis; as yet insufficiently studied, but united to a former class by a mere pathogenic parentage. And thirdly, we have the vascular cirrhosis. In hypertrophic cirrhosis with icterus surgical interference is indicated in the absolute failure of medical treatment. I believe that physicians generally admit that in this affection the prognosis is unusually serious, and that although the evolution of the process is ordinarily quite slow, all cases observed up to the present time have ended fatally. Consequently, when these patients are left to themselves, they are condemned to death, because no matter what medical treatment may be followed, even for a long time and under the best conditions, it is absolutely powerless to put a stop to the ever progressing evolution of hepatic sclerosis. Secondly, surgical interference is absolutely benign, and in this respect I would recall the opinion of Terrier, who says: "Il est, en effet, certain que l'exploration du foie, après incision sur la ligne médiane, est une chose possible, presque facile et d'une innocuité indiscutables des qu'on utilise la méthode antiseptique, soit même la méthode mixte à la fois aseptique et antiseptique." And still more, if one takes into consideration the operative results of cases so far reported, it will be found that the operative mortality has been excessively low. I believe, for all these reasons, that a well-conducted cholecystostomy is practically devoid of any operative gravity, and the patients submitting to this operation run little or no risk. Thirdly, we have the happy results obtained by surgical interference, and the question comes up as to whether or not the operative cure has given rise to therapeutic successes, both immediate and definitive. Without relating the many brilliant recoveries obtained by Terrier, Michaux and Delagenière, and others, I will merely add three of my own, which practically represent what others have experienced.

M. H—, a young man æt. 24 years, had always enjoyed excellent health up to the summer of 1901. At this time he commenced to experience the symptoms of acid dyspepsia accompanied by attacks of gastric pain. For five months these became more and more frequent and were frequently followed by icterus, while the general condition of the patient became poor. During the attacks of pain it appeared as if the gall-bladder was the seat of the trouble, but finally it extended over the liver These attacks of pain and the icterus made me suspect that the patient was afflicted with gall stones, although a careful examination of the stools after these attacks failed to reveal the presence of even the minutest calculus. As the patient was be-

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coming worn out by constant pain, and was losing flesh rapidly, operation was decided on in November. Examination of the patient at this time showed that he had slight icterus, with an evening temperature of 38·2° C. Digestion was difficult, the stools clay-coloured, and the urine containing biliary pigment, but no albumen. By percussion the liver extended two fingers' breadth below the ribs, and palpation of the gall-bladder was painful. The heart and lungs were normal. On November 27th the abdomen was opened in the semilunar line and the gall-bladder was found buried in quite dense adhesions, but careful exploration failed to reveal the presence of calculi, either in the gall-bladder or in the ducts. The liver was enlarged, but regular in shape. After walling off the cavity, the fundus of the gall-bladder was incised, giving exit to a dark, purulent bile, and the finger introduced within its cavity showed that no calculus was present. The gall-bladder incision was enlarged, and the walls were found thickened and soft, and after having carefully explored the under aspect of the liver and ducts for the second time, cholecystostomy was decided upon, a large drainage-tube being introduced into the organ. The results of this operation were perfect; there was no rise in temperature after the operation, and on the second day the dressings became soaked with bile, and at the same time the patient expressed himself as feeling greatly relieved. The drainage-tube was allowed to remain for seventeen days, at which time the bile had taken on all its natural attributes, the icterus had completely disappeared, and the patient digested everything he ate perfectly. The drain was consequently removed, and the patient was discharged in excellent condition with the biliary fistula closed twenty-seven days after the operation. Bacteriological examination of the bile taken at the time of the operation showed the presence of the *Bacterium coli* and the *Staphylococcus aureus* in large numbers.

I have followed this case since the operation, and the patient has remained in perfect health ever since.

The second case was that of a young woman, æt. 27 years, who gave the following history: She had been married two years and had had one labour at term, which presented no complications. Five years ago typhoid fever, apparently rather severe. Menstruation always regular and painless. No history of biliary calculi. I suspected, from certain things, that the patient was addicted to the use of alcohol. When the patient came under my observation she had been suffering from pain in the region of the gall-bladder for some nine months, and had recently become quite cachectic. She was a frail woman and complained constantly of a pain in the pit of the stomach, and which extended to the right side. Examination of the thorax was negative. The urine was scanty, averaging about 750 c.c. in twenty-four hours, having a slightly acid reaction and a deep orange hue. Examination showed that it contained slight traces of albumen, a certain amount of pus, and bilirubin.

Upon physical examination of the abdomen, the region of the liver felt tense, and this apparent tumefaction extended to a little beyond the median line. It was hard and appeared somewhat movable, but was in no way connected with the genital organs, although it extended nearly to the right iliac fossa. As I could not make up my mind as to the diagnosis, an exploratory incision was advised and accepted. An incision was carried from the costal border to below the umbilicus in the semilunar line, and when the peritoneum was opened the tumour was found to be nothing more than a greatly enlarged liver. The shape of the organ had, however, been preserved. The gall-bladder was exceedingly enlarged and sausage-shaped, but no calculi could be felt either in the gall-bladder or the ducts. By raising up the border of the liver with the hand inserted deep into the abdomen it was an easy matter to bring the gall-bladder into the abdominal incision. On account of the apparent changes which the hepatic parenchyma had undergone, as well as the condition of the gall-bladder, I decided to do a cholecystostomy followed by drainage of the gall-bladder. This was carried out according to Delagenière's technique, which I will describe further on, but was somewhat difficult of execution on account of the projection of the liver-border.

The temperature, which had oscillated around 39° C. before the operation, dropped to the normal on the second day, and for the first four days following the interference a large amount of mucous was discharged from the gall-bladder. Bacteriological examination showed that this fluid contained the bacterium coli in large numbers. On the fifth day, however, the fluid from the drainage began to

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take on the appearance of bile, and during the next four days it gradually transformed, until nothing but pure bile was found on the dressing. The patient rapidly improved, her digestion became perfect, and the temperature remained normal. The tube was removed on the twentieth day, after which the fistula rapidly closed, and the patient was discharged in excellent condition thirty-seven days after the operation. She was ordered a strict milk diet, which was continued for three months, after which time she was completely cured, and, as far as my knowledge goes, has remained so ever since.

My third case may be briefly summed up as follows: The patient, a male æt. 56 years, was referred to me for chronic jaundice and occasional painful attacks in the region of the liver, accompanied by a rise in temperature, this condition of affairs having existed for fourteen months. The abdomen was considerably distended with fluid, the patient stating that the abdomen had continued to enlarge for the past three months. The general condition was very poor, the patient having lost considerable flesh; the liver extended three fingers' breadth below the costal border, and with some difficulty indications were noted pointing to enlargement of the spleen. The abdomen was opened, and a large cirrhotic liver was exposed, with evidences of chronic peritonitis in the infra-hepatic region. The gall-bladder was greatly enlarged, its walls thickened, but no calculi could be found within it or in the ducts. The ascitic fluid having been let out, a cholecystostomy with drainage was done. After the operation the temperature remained at normal; the tube was removed at the end of one month, after which the resulting fistula permanently closed in about a fortnight. The bile obtained at the operation contained *Bacterium coli*. The icterus disappeared, the appetite came back, and the patient made weight. Four months after the interference the general condition remained excellent, but the fluid had again made its appearance in the abdomen, so that puncture became necessary, removing 7 litres of a dark-yellow, thick fluid. After this the patient remained well. I had news of him a year afterwards, at which time he was fairly well, there being no trace of jaundice, and, on the whole, he appeared well pleased with his condition. However, the advanced stage of the hepatic process will naturally not allow a permanent cure to be obtained in this case, and it is evident that the patient will ultimately die of the process, but the operation certainly greatly improved his condition and without doubt gave him a lease of life of several years.

The majority of the considerations I have put forward relative to the prognosis and the ineffectiveness of medical treatment may also be applied to cases of hypertrophic cirrhosis without icterus, and in looking over the reported cases it is most curious to note that the results obtained by surgical interference have been very good, mostly resulting either in a radical cure or a very evident improvement. As to vascular cirrhoses, we may say in a few words what we have already indicated relative to hypertrophic cirrhosis with icterus, namely that medical treatment is powerless to stamp out the process of sclerosis, although it should be remarked that contracted livers are less amenable to treatment than the large liver, and that the process inevitably terminates in death. That something may be gained by operative treatment in these cases I believe is logical to suppose, and several cases have been reported where simple laparotomy has been sufficient to cause the ascites to disappear, likewise the cirrhosis itself in a few cases. Instances of this kind are numerous, but, having no personal experience, I will relate as an example one reported several years ago by Routier, where the patient was supposed to have a tuberculous peritonitis, and an exploratory incision showed that in reality an atrophic cirrhosis was present, but the interesting part of the case is that the exploratory incision, followed by a few punctures for the ascites afterwards, cured the patient in three months. In one of Delagenière's cases the question might arise whether it was the laparotomy or the drainage of the gall-bladder which caused the favourable result, but my own opinion is that the biliary fistula contributed greatly to the improvement of the patient's condition. Since the ascites did not recur, it is probable that the drainage of the gall-bladder favourably influenced the phlebitis and periphlebitis of the portal and infra-hepatic veins, and, so to speak, corrected the intra-hepatic circulation, and, on the other hand, since the icterus diminished, it is to be supposed that the drainage of the gall-bladder had a fortunate action on the angiocholitis. However, we cannot draw any conclusions from a single case, and consequently it is only future experience which will teach us the truth.

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Relative to the technique to be employed for draining the septic biliary tracts, one may choose between cholecystogastrostomy, cholecystenterostomy, and cholecystostomy. The first two do not appear to be applicable to the cases we are considering. The first is a very exceptional operation, having special indications, while the second has the great inconvenience of turning back the infectious elements contained in the bile into the organism by throwing it off into the intestine. On the contrary, in cholecystostomy we have an ideal operation. The cutaneous fistulisation of the biliary tract gives exit to the infected bile, and from this fact the biliary tract becomes thoroughly drained, even in its most remote intra-hepatic ramifications, and consequently disinfection of the liver is admirably realised. The large majority of authors accept this way of thinking, and consequently this operation is currently practised at the present time, but there is certainly a choice of procedure, because the classical cholecystostomy presents certain disadvantages. Without doubt the permanency of the fistula is the most serious objection, because it gives exit to a certain amount of bile continually, and not infrequently requires a second interference for its closure; and although there are numerous instances of patients who enjoy a satisfactory condition of health in spite of this loss of bile, it nevertheless is incumbent upon the surgeon to select a technique which will result in a temporary fistula. For this reason Terrier believes that one should make the fistula as small as possible, in order to avoid a too abundant flow of bile, and more especially in order to make the occlusion of the fistula an easy thing to do. For the same reason Delagenière has worked out a technique by drawing the gall-bladder through a button-hole made in the right rectus, so that the fistula will be more continent and more easily allow its closure. The opening of the gall-bladder is consequently furnished with a muscular sphincter, which holds the borders in contact as soon as the drainage-tube has been removed. This change in technique has resulted in the formation of a true temporary cholecystostomy, the technique of which I will rapidly describe.

The incision is carried along the external border of the right rectus and in length should be about 12 cm., a space which is usually sufficient for the introduction of the hand. Its starting-point is at its costal border. Naturally the cutaneous incision should be longer than that of the aponeurosis and of the muscle, and the aponeurotic incision should be longer than that made into the peritoneum. In proceeding in this way there are many advantages which at once become evident and need no discussion. When the peritoneum is opened the gall-bladder is packed around with gauze sponges so as to prevent contamination of the abdominal cavity with bile, the gall-bladder is drawn up, and the fundus incised. After the bile has escaped the interior of the organ is explored with the finger, and the field of operation being freely irrigated with salt solution in order to remove the septic contents of the gall-bladder, and the gauze compresses having been changed, the formation of the biliary fistula is proceeded with. At about 15 or 20 mm. from the borders of the incision in the fundus a series of sutures are placed, including only the muscular layer and the peritoneum. The loops of these sutures are brought together, but are not interlocked. The gall-bladder is then ready to be stitched in place. A running suture closes the peritoneum above the gall-bladder, and then the two ends of each suture are passed through the neighbouring parietal peritoneum. When this is done the peritoneum below the gall-bladder is closed with a running suture, and nothing more remains than to tie each suture in the gall-bladder separately. The top of the gall-bladder which has thus been sutured to the parietal peritoneum projects into the wound, but this projection need not be greater than that necessary to protrude through the rectus muscle. A button-hole is made parallel to the fibres of the rectus at about 5 or 6 mm. from its external border, and through this muscular button-hole the fundus of the gall-bladder is pulled with a pair of forceps, and then the borders of the opening are united to the anterior aponeurosis of the muscle by interrupted sutures, which should include the entire thickness of the wall of the gall-bladder. Should it be found that the opening into the gall-bladder is unnecessarily large, it may be lessened by a few interrupted sutures. A drainage-tube which exactly fits the opening in the fundus of the gall-bladder is then introduced. The closure of the abdominal incision should be done as in any other laparotomy.

The after-treatment in this operation is usually simple, and after the drainage-tube has been removed permanently the fistula is allowed to close itself, a thing which usually takes place within the following week or fortnight. The advantages of this operative procedure appear to me real, because the operation is easily performed. However, the most evident advantage that it presents, according to my way of thinking, is that the fistula closes almost immediately after the drainage-tube has been removed. The time at which the tube should be permanently removed will vary considerably according to the condition of the gall-bladder found at operation, but usually the condition presented by the bile will allow one to dispense with the drainage from a fortnight to three weeks afterwards.

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A CLINICAL LECTURE

ON

A GROUP OF CASES ILLUSTRATING THE SURGERY OF THE BILIARY APPARATUS.

By ARTHUR E. BARKER, F.R.C.S.,
Surgeon to University College Hospital.

GENTLEMEN,—When called upon to operate for affections of the gall-bladder many considerations present themselves to the mind of the surgeon even when he feels fairly sure of the diagnosis. I hope that the cases briefly sketched in the following group, which have all been under my care here or in private comparatively recently, may assist some of you to form clear ideas as to the line to be taken in such cases.

Of course the first consideration is the diagnosis, which you must already know is not always so easy a matter as is often supposed. Two cases are included in this group which turned out to be unconnected directly with the biliary apparatus but which had several features about them suggesting that such a connection existed. They are introduced partly because of this and mainly to remind you that difficulties of diagnosis will arise.

Then we come to the question how best to reach a part of the body which in the case of the biliary ducts is most inaccessible. To reach the gall-bladder alone is very easy as a rule if it is of about normal size. But if it be small and shrunken, as it so often is, and especially if the liver is large and the patient is obese, this is anything but the case. As a rule a vertical incision along the outer border of the rectus muscle for about four inches, commencing about one inch below the border of the ribs, is sufficient for all the ordinary operations on the gall-bladder. But for the more inaccessible cases and for those in which the common duct has to be dealt with something more must be done.

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Here the first incision may be prolonged at its upper end inwards and upwards across the rectus as was done in some of the cases to follow, or the first incision of all may be made obliquely upwards and inwards about an inch from the costal cartilage border. Both of these, if long enough, give free access to the parts in question. But it is obvious that to cut across the muscles is undesirable. For this reason in all cases where it is adequate I prefer the vertical incision alone. I have never done the single oblique trans-muscular incision and have only added a short oblique incision to the vertical in a few cases, as you will see presently.

Then the finding of the common duct is one of the difficult problems which confronts us. In the cases in question I have followed the practice which is adopted by many of slitting up the cystic duct until it reaches the ductus communis choledocus. This appears to me an excellent routine from which, of course, we may and must depart in special circumstances.

In only one of the following cases was the common duct when opened subsequently stitched up again. Reliance was placed upon drainage for several days until the healing process could be left to itself.

In only one case was it necessary to open the duodenum to gain access to a calculus jammed in the ampulla which in its turn was imbedded in a mass of adhesions.

These points you will find illustrated in the following cases, and their practical bearing will be clear in each, I hope. One thing is fairly certain in this branch of surgery, viz. that no hard and fast routine can be followed in all cases.

Case 1.—Gall-stone impacted in the common duct. Cholecystectomy. Common duct explored from duodenum. Stone removed. Recovery.

D. C—, æt. 59 years, was operated on by myself three years ago for symptoms which pointed as far as could be made out to duodenal ulcer leading to stenosis. Gastro-jejunostomy was performed and much scar-tissue was found about the second part of the duodenum. Soon after convalescence, which was uneventful, the man suffered from an attack of jaundice, but this did not last long. Two years later he was treated in the medical wards for jaundice again, and as the usual measures gave no permanent relief, he was sent to me for operation. On opening the abdomen the gall-bladder was found in a mass of adhesions and much atrophied. Stones were felt in the common duct, which was slit up from the gall-bladder for their removal. But it was impossible thus to remove one larger one, jammed in the ampulla. The second part of the duodenum was therefore opened and with the forefinger in the gut and the thumb on the stone within the duct, I was able by enlarging the opening in the latter to remove the mass. The gall-bladder was then excised and a drain-tube passed down to the duct, which was left open. Gauze packing completed the very difficult and prolonged operation. The patient suffered a good deal for the first few days, but recovery was uneventful and the motions soon resumed their normal colour.

The question whether the duodenal stenosis requiring gastro-jejunostomy was in the first case due to cicatrisation round an inflamed gall-bladder or to an ulcer in the duodenum, as was supposed, has now to be reconsidered. Since the operation I am inclined to think it was the former. No case could have better illustrated the difficulties surrounding such an operation, here happily surmounted, but demanding much deliberation and some loss of time, with consequent risk to the patient.

Case 2. — Cholelithiasis ; cholecystectomy ; recovery.

Mrs. B. B—, æt. 38 years, had had a typical history of attacks of biliary colic many times during the last fifteen years, but no jaundice. Neither the liver nor gall-bladder could be felt. The attacks had grown more severe. A longitudinal pararectal incision was made, and the gall-bladder was found full of stones, but shrunken. To reach it fully the incision had to be enlarged inwards and upwards obliquely across the rectus. The gall-bladder was then stripped from the liver, the cystic duct tied and cut through, and the abdominal wound closed. This was a typical operation, the anatomical details being particularly well seen, and was the only one in which drainage could be dispensed with. The wound healed *per primam*, and convalescence was quite satisfactory.

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CASE 3.—Suppurative cholelithiasis; *B. coli communis*. Abscess of liver under gall-bladder and reaching to portal fissure. Abscess opened and stitched to abdominal wall. Gall-bladder opened separately, muco-pus evacuated, and several stones drained separately. Death a month later.

Mrs. W—, æt 40 years. History of "colic" twenty years ago, and for the last nine weeks had had pain in the right hypochondrium and in mid-line after food, which was frequently vomited three hours afterwards. There was resistance to palpation, felt deeply in the region of the gall-bladder. This was ill-defined except on the right side, where it felt hard and irregular. It moved with respiration. The rest of the liver was not enlarged. Otherwise the abdomen was normal. Temperature 102° F. Respiration 39° F. Pulse 128° F.

Operation, vertical pararectal incision. Swelling seen to be the gall-bladder projecting below the liver and welded to the latter by a mass of inflammatory tissue under which was fluctuation. During manipulation the surface of the liver close to the gall-bladder gave way and a large hepatic abscess discharged. When emptied the edges of this abscess were stitched to the abdominal wall. The gall-bladder was now separately opened and cloudy muco-pus evacuated together with several calculi. The cystic duct appeared to be obliterated. The opening of this viscus was separately stitched to the peritoneum, a gauze drain being placed within the bladder and a rubber tube in the hepatic abscess, which reached deeply. The rest of the wound was closed.

For three weeks all went on very satisfactorily; the temperature was normal and the drainage good. I left town for a holiday feeling quite easy about her. Soon after she began to vomit frequently and the pulse became poor. Each day she became weaker without any rise of temperature until a week later, when shortly before death it rose to 104·6° F. There was no distension or tenderness and sickness had ceased. She died exhausted in the fifth week. No peritonitis was found, but a large abscess in the liver was still unrepaired and reached to the deep part of the organ.

CASE 4.—Suppurative cholecystitis and gall-stones; cholecystostomy; drainage; recovery.

Mrs. B—, æt 46 years, admitted for abnormal vomiting of three days' standing, and very severe pain in the right hypochondrium, marked constipation, no jaundice.

Operation, pararectal vertical incision. Adhesions round the gall-bladder uniting the liver to parietal peritoneum. Gauze packing and puncture of the gall-bladder. At first mucus escaped, then pus. Gall-bladder opened widely and five calculi of large size removed. The edges of the hole were stitched to the parietal peritoneum round a drain-tube which led into a bottle. Rest of the wound closed. Tube removed on the fifteenth day. Convalescence uninterrupted.

CASE 5.—Cholelithiasis; cholecystitis; cholecystectomy without opening gall-bladder; recovery.

Mrs. B—, æt. 28 years, lately in fever hospital for enteric of a mild form. Typical Widal's reaction. Three weeks after leaving attacked with pain in right hypochondrium with fever. On admission a mass was felt there which soon subsided, as also the fever. This condition fluctuated for nearly a month in the medical ward.

Operation: Vertical pararectal incision. Gall-bladder distended, inflamed, thickened, removed without being opened. When stripped as far as the cystic duct the latter was found to be solid and broke short off through the closed portion. No fluid escaped from either stump and no ligature was required. Gauze drain and partial closure of the wound. The gall-bladder, as large as a medium-sized pear, was very thick-walled, and contained four large stones, which had caused extensive ulceration of the inflamed lining membrane. Convalescence was uninterrupted.

CASE 6.—Carcinoma; nodule involving the common duct; extreme jaundice and suffering. Cholecystenterostomy; complete relief.

Thomas E—, æt. 67 years, admitted under the care of Sir Thomas Barlow for intense jaundice, and a large tumour reaching from the liver to below the spino-umbilical line, evidently the distended gall-bladder. The liver also was obviously much enlarged. Diagnosis: Carcinoma of head of pancreas involving the common duct. As the suffering was very great from the icteric pruritus the patient was transferred to my care for operation, which he greatly desired. This was undertaken with some misgiving, owing to the patient's age and feeble condition.

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Operation: Vertical pararectal incision. Gall-bladder found about 6 × 3 inches in size, smooth and distended with thin green bile. It was turned out of the wound and evacuated. When pulled up, a white nodule of growth was seen involving the common duct about the size of a walnut. The gall-bladder was now anastomosed with the upper part of the jejunum by a fresh opening about one inch long, and the first opening in the gall-bladder was turned in and stitched up. The abdominal wound was then closed without drainage. Primary union followed without interruption, and convalescence was uninterrupted, the patient going home on the thirty-first day. It was very remarkable how soon the intense jaundice disappeared and the consequent distress. A letter was afterwards received giving a favourable account of the patient's general condition. He lived some months in comfort and then died of asthenia, due to the cancer.

Case 7.—Cholelithiasis with many adhesions; cholecystectomy; recovery.

E. W—, unmarried woman, admitted for severe pain and a tender mass in the right hypochondrium and fever (101°.) The temperature had been higher before admission. The tumour had all the characteristics of a distended gall-bladder in a state of inflammation. There was slight jaundice and the liver was a little enlarged. After some days in hospital the general condition improved and the swelling decreased.

Operation: Vertical pararectal incision. Gall-bladder inflamed, distended, and welded to adherent omentum. While separating these adhesions a weak spot in the viscus gave way, but the opening was at once clamped. It was then separated from the liver and removed after ligature of the cystic duct. Some troublesome bleeding took place from the raw surface of the liver, which could not be stopped by ligature or cautery, but was at once arrested by the injection into the adjacent hepatic tissue of a few c.c. of adrenalin chlor. solution, 1 in 40,000, which blanched the whole raw surface at once. The omentum was then packed round the area of operation, and a gauze drain was introduced down to the cystic duct stump, the abdominal wound being otherwise closed.

The gall-bladder was found to contain a lobulated calculus jammed in the cystic duct, and was much inflamed and ulcerated in patches. Convalescence was uninterrupted, the patient going home in the fifth week.

Case 8.—Suppurative cholelithiasis. Cholecystectomy after slitting up common duct. Recovery.

Mrs. G—, æt. 52 years. Many attacks of biliary colic accompanied by jaundice during the last eighteen months; the most recent was five weeks ago, and, like those before it, was accompanied by vomiting. On admission a small tumour was felt at the seat of the gall-bladder, but the liver was apparently normal.

Operation: Vertical pararectal incision, afterwards carried upwards and inwards obliquely across the rectus a couple of inches from the costal margin. No adhesions about the gall-bladder. The latter was packed with gauze, opened, and a large number of stones removed. The common duct was then slit up and a probe passed through it into the duodenum, and also up the hepatic duct, no calculi being found in either. The gall-bladder was then excised after tying the cystic duct. The common duct was left open with a drain tube carried down to it. A gauze apron stuffed with iodoform gauze covered this. These were removed on the third day, and the apron on the fourth day. Convalescence was uninterrupted, the patient going home in the fourth week.

Case 9.—Symptoms resembling severe biliary colic; exploration; hard mass in second part of duodenum with surrounding adhesions; ? duodenal ulcer; gastro-enterostomy; recovery.

C. D—, æt. 64 years, looking younger. Severe epigastric pain for two years, in attacks growing gradually worse, especially for the last two months. This pain begins in region of gall-bladder, spreads to the right under the shoulder. Occasional vomiting; no blood; no jaundice; no gall-stones passed; stomach slightly dilated; liver not enlarged.

Operation: Vertical pararectal incision, afterwards prolonged obliquely inwards and upwards one and a half inch from costal margin. Gall-bladder distended, but empties under gentle pressure; no calculi or other abnormality. A

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hard mass the size of half a walnut in the first or second part of the duodenum, with a small, movable gland in the gastro-hepatic omentum. As this mass was inoperable owing to its relations to the common duct and vessels, I at once did a retro-colic gastro-enterostomy to give the part rest and to meet the obstruction present and to come. The presence of inflammatory adhesions about the mass suggested a simple chronic duodenal ulcer. The gastro-enterostomy was easy in spite of the incision being outside the right rectus.

Convalescence was perfectly smooth. A subsequent analysis of a test meal showed great diminution of hydrochloric acid, so that we cannot exclude cancer in this case.

CASE 10.—Symptoms obscure, rather pointing to cholelithiasis; hard lump and adhesions in second part of duodenum; probably chronic ulcer; gastro-enterostomy; recovery.

A. D—, æt. 46 years. History of vomiting dark material on many occasions since twelve years ago, when the first attack began with copious hæmatemesis. There were also "tarry" motions. Pains in the stomach have been complained of during the same time. Vomiting during the last year has been two or three times a week and the ejected matter coloured like chocolate, and frothy. There has also frequently been dark or bright blood in the motions. Piles were also complained of. The character of the pains and the history seemed to point either to chronic duodenal ulcer or to cholelithiasis.

Operation: Vertical pararectal incision. A hard mass was found in the second part of the duodenum surrounded by many ordinary inflammatory adhesions: there were no gall-stones or inflammatory changes in the gall-bladder. A retro-colic gastro-enterostomy was performed through the first incision and was not particularly difficult.

Convalescence was rapid and all the trouble was relieved.

CASE 11.—Large stone in common duct; removed by choledochotomy; gall-bladder excised; subphrenic abscess opened between ninth and tenth ribs; recovery.

An unmarried lady, æt. 50 years, had had attacks of biliary colic on and off for years, accompanied by slight jaundice. No definite swelling. The patient was very thin and constantly ailing, Dr. Lynch and Mr. J. P. Bartlett regarding her condition as due to gall-stones.

Operation: Incision as before; gall-bladder distended; it was stripped from the liver and opened, the slit being caried forwards into the common duct, which was found distended to a size at least equal to that of the duodenum, for which it was at first mistaken. From it was removed a spherical calculus as large as a small grape. The gall-bladder was then removed and the common duct stitched up. A gauze apron was laid into the wound and then packed with iodoform gauze, and the omentum arranged outside of this. The abdominal wound was then partially stitched. On the third day the inner packing was drawn out and much turbid serum followed. After this the temperature rose, and a few days later symptoms of subphrenic abscess on the right side appeared. This was first emptied by aspiration in the ninth interspace behind, and next day by free opening. At this time the lady was very ill, with high fever and delirium, which lasted a couple of weeks, and then gradual improvement took place and both wounds closed. The patient is now in excellent health, better than she has enjoyed for many years.

CASE 12.—Gangrenous cholelithiasis; large stone impacted in cystic duct; stone removed; gall-bladder drained; recovery.

Mrs. I—, æt. 33 years. This lady had had two or three attacks of biliary colic at intervals of some months, for which she was treated with the best results by Dr. Holman, of Hampstead. The last attack, for which I was consulted, together with Drs. Holman and Newton Pitt, was very severe, and accompanied by high fever and vomiting. There was only a little jaundice. A large swelling could be felt in the region of the gall-bladder, and this was very tender.

Operation: Vertical pararectal incision. The gall-bladder was at once seen much distended, of a deep purple colour, flecked with grey patches, evidently gangrenous. It was imbedded in adhesions with the omentum, colon, and stomach. Some of these were cautiously separated and a gauze packing was placed round the gall-bladder. An attempt was then made to draw the latter forward, but one of the patches gave way, and

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the inflammatory fluid began to ooze through. The opening was at once enlarged and the contents mopped up quickly. One very large calculus was now extracted, which was impacted in the cystic duct. It was found to be the size of a large date and was more or less conical, but without facets. The gall-bladder was so rotten that all stitching was out of the question, and the fluid coming from it suggested the presence of cholangitis. It was therefore drained without further interference. The packing was changed, and after careful revision of the whole area of operation a rubber tube was fixed in the gall-bladder and the wound around was packed with gauze. The abdominal incision was partially closed. The dressings were changed in a few days, the bile all the time draining into a bottle at the bedside. No complication followed, and convalescence was unexpectedly rapid.

CASE 13. — Suppurative cholelithiasis; large stone in cystic duct; removed; gall-bladder stitched up; wound drained; recovery.

Unmarried lady, æt. 64 years, had had several attacks during the past year of pain in the epigastrium, and on one occasion (July, 1905) this was accompanied by much swelling in the right hypochondrium, with high fever. When I first saw the patient there was no swelling and only a trace of tenderness and resistance. There was no fever and the lady's condition was good. The diagnosis of those who had previously seen her was "carcinoma of the gall-bladder," this being supported by the presence of a definite tumour and a very strong family history of cancer. My own diagnosis was cholelithiasis; this was supported by Sir Thomas Barlow.

Operation: Vertical pararectal incision. Gall-bladder surrounded by adhesions and much thickened, containing muco-pus. One large calculus was impacted in the cystic duct, which was so enormously thickened round it as to suggest carcinoma. The hard, white mass here formed was incised and a slice removed for examination. There were some enlarged glands in the fissure of the liver, but they were soft as if only inflamed. Both incisions in the gall-bladder were stitched after removal of the stone, and a gauze packing completed the operation. Drainage was good and convalescence uninterrupted.

Microscopical examination showed the portion of cystic duct removed to consist solely of inflammatory tissue.

CASE 14.—Cholelithiasis; cholangitis; cholecystectomy and drainage of common duct; death.

H. C—, æt. 53 years, suffered four years ago from an attack of pain in the region of the gall-bladder, with jaundice, which lasted a week. At the beginning of December, 1905, a similar attack was experienced, with severe pain. The jaundice followed four days later and lasted up to admission, on December 15th. By this time the pain had subsided to a dull ache in the epigastrium, but the jaundice persisted in a marked degree. The liver could not be definitely felt and was not tender.

Operation: Vertical pararectal incision, subsequently extended obliquely across the rectus one and a half inches from the costal border. The gall-bladder was shrunken under the liver and rotten. During manipulation it gave way, part of it, in a state of ulceration, remaining incorporated with the liver. A number of small calculi were removed with a scoop. The rest of the bladder was freed, tied at the cystic duct, and removed, together with several good-sized stones.

The common duct was then opened and found to contain many calculi, some of which extended into the hepatic duct. All these, which could be felt, were removed and a probe passed into the duodenum. A rubber tube was placed in the common duct and led out of the wound, and round it was placed an apron of gauze, within which was a packing of iodoform gauze. The strength of the abdominal muscles rendered this operation one of great difficulty. The two first days after operation the patient seemed fairly well, but on the fourth the pulse and breathing became rapid, the temperature rose to 103·8° F., and he died on the evening of the fourth day, the jaundice having deepened.

The following abstract of the pathologist's more extended notes of the post-mortem examination are copied from the report of the case:

"General peritonitis found; intestines distended and adherent by soft yellow lymph. Site of removal of the gall-bladder quite healthy. Common hepatic duct dilated; former contains several small stones and a small amount of bile-stained muco-purulent material. Smaller ducts show thickening. No

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abscesses in the liver, which is large and bile-stained, and markedly fatty. Spleen large and very soft. Kidneys small, granular, with small cysts. The right has a large cyst at its upper end, capsule adherent. Arteries much thickened; vessels atheromatous. Heart hypertrophied; other organs normal."

CASE 15.—Cholelithiasis; cholecystectomy after freeing adhesions; recovery.

Mrs. S—, æt. 68 years, hale but showing a markedly rickety skeleton, was admitted under Dr. Martin for pain and tenderness in the region of the gall-bladder which resisted medical treatment. There was an indistinct hardness under the liver. No jaundice at any time.

Operation: Vertical pararectal incision. The gall-bladder was adherent to the duodenum and in separating it a small hole was torn in it but immediately clamped. The viscus was rapidly separated from the liver up to the cystic duct, which was tied and cut through with Paquelin's cautery. It was thickened and contained calculi. A gauze apron was then arranged over the stump, packed with iodoform gauze strips, and the wound in the abdominal wall partially closed. The patient bore the operation well. The inner packing was removed on the third day, the apron on the fifth, and the wound closed rapidly. Convalescence was absolutely uninterrupted.

CASE 16.—Cholelithiasis and cholangitis; cholecystectomy and opening of the common duct, which was drained with a rubber tube; recovery.

E. S—, unmarried woman, was admitted with high fever (104° F.) and jaundice, with pain in lower part of right side of the chest and under the shoulder blades for two days, during which she was without sleep. Similar attacks for about one year but worse for the last five months since rheumatic fever. Each attack, of which she had had about six in five months, lasted three or four days and had been accompanied by rigors and vomiting. The bowels were regular, the stools light coloured or actually white and not offensive.

Operation: Vertical pararectal incision. Gall-bladder swollen, thickened, and inflamed. It was too friable to remove from the liver entire and was first evacuated. It was then stripped from the liver, during which procedure the raw surface of the latter bled rather freely. This was stopped by injecting into the tissue under the wounded surface about 10 c.c. adrenalin chloride solution, 1 in 40,000, the effect of which was to cause blanching of the contiguous hepatic tissue and speedy cessation of the oozing. When the gall-bladder was quite separated from the liver up to the cystic duct the latter was tied close to its termination. But before it was severed it was used as a guide to the common duct. This was opened and explored for calculi. None were found either in it or in the hepatic duct, which were freely searched upwards and downwards with a probe, as far in the latter case as into the duodenum. The cystic duct was now divided and a small rubber drain-tube was inserted well into the common duct and led out of the abdomen. Round this a gauze apron was adjusted and within the apron a stuffing of iodoform gauze strips. The abdominal wound was partially closed. As there was a strong suspicion of cholangitis the drainage of the tube in the common duct was anxiously watched. The bile ran off through it freely and was soon normal. After three days the inner packing was drawn out and on the fifth the apron. The tube was left a few days later and as it seemed to produce very little bile it was taken out. It was obvious that the bile was running its usual course now into the duodenum. After this there is nothing to record but a smooth convalescence.

CASE 17. — Chronic icterus, enlarged "hob-nailed" liver, and wasting. This last case, whom I have seen to-day convalescent, and who returns to his home to-morrow well, is of particular interest. A gentleman, æt. 76 years, consulted me five weeks ago on account of jaundice well marked for seven months, with great wasting, an enlarged liver, and great depression. To be brief, the question had to be considered whether this was a case of carcinoma in or about the under-surface of the liver or a gall-stone impacted in the common duct. The evidence was about evenly balanced; and in view of the patient's age, the fact that he had a very irregular heart, and that during his attacks of pain he had been very collapsed, I negatived operation, as it happened unwisely. A few days after returning home he wrote to me saying that he wished an operation done to settle the

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matter one way or the other. I therefore operated on this patient, though with much misgiving. There is no need to go into details. I found a shrunken gall-bladder, full of calculi, a "hob-nailed" liver (the patient was perfectly temperate), and stones in the common duct. With great difficulty the duct was cleared and a gauze drain and tampon were inserted.

From the small amount of bile seen when the ducts were opened I feared that the liver was so disorganised from chronic interstitial hepatitis, due to the cholecystitis and cholangitis, that it might possibly fail to resume its functions. For the first few days this fear seemed justified, but a few days later the motions resumed their normal colour and the urine became pale. From this on all went well. He leaves the Nursing Home to-morrow with only a trace of jaundice. This is probably due to the remains of bile-pigments in the tissues, where they have been for at least eight months. This was the most difficult operation of the kind which I have ever performed. But it caused no shock and recovery was quite without incident.

Glancing back at this series of conditions involving the biliary apparatus, all more or less occurring within a short period, the considerable variety they offer, both in the conditions met with and in the means required for dealing with them, is worth noting.

In the first place, let us turn to the fatal cases, two in number, and learn what we can from them. The first (Case 3) illustrates the evil effects of delay in some cases of cholelithiasis. Here the inflammation of the gall-bladder had been allowed to become chronic, and as a consequence a gradual extension of the infection to the liver until a large ragged abscess in its substance was produced. This by common consent of all surgeons of experience is a most dangerous condition even when freely opened and thoroughly drained as in this case. For not only is there the great danger of infection of the peritoneum (happily surmounted in this case) but there is the danger of pylephlebitis at any time in the course of the subsequent drainage or of exhaustion, which was here fatal. The patient did well for three weeks, the temperature being normal. Then on the twenty-first day she began to vomit constantly, and though there was no fever for the next few days the vomiting persisted. Then on the twenty-ninth day the temperature began to rise and she died with 104·8° F. A large ragged abscess was found in the liver extending to the portal fissure but nothing more. There was no peritonitis and the gall-bladder had continued to drain perfectly. Our only regret was that the patient had not been operated on early enough, but over this we had no control.

The next case (No. 14) illustrates the dangers of cholangitis, a condition recognised by all as grave in an extreme degree, and almost always beyond the reach of surgical help when fully developed. It is intrinsically dangerous as a septic infection of the biliary passages, and as likely to lead on to abscess or pylephlebitis, and it is specially dangerous during operations for its relief on account of the difficulty of draining the hepatic ducts without infecting the peritoneum. This can sometimes be done where there are no calculi in the common duct by bringing the gall-bladder to the surface and fixing it there, and opening it for drainage on the surface of the body. But this could not be done in this case. There were calculi impacted in the hepatic and common ducts which had to be removed. And though this was done and the ducts were efficiently drained, infection of the peritoneum took place and the cholangitis persisted. A combination of these two conditions led to the patient's death on the fourth day. There is every reason to suppose that an operation some weeks earlier would have saved him.

The only other case which gave rise to anxiety was No 11. Here the danger of another form of infection was well illustrated. A right subphrenic abscess developed soon after operation and only prompt action saved the patient's life.

Two other cases, it will be noticed, illustrated conditions which might be easily taken for trouble primarily in the biliary passages. In both a tumour was found in the second part of the duodenum, close to the common duct. Both were relieved of all their symptoms by immediate gastro-enterostomy as soon as exploration had revealed the nature of the condition.

The general line of action in all the cases of cholelithiasis, it will be seen, was this: Where the gall-bladder was deeply inflamed and there was a suspicion that cholangitis existed too it was simply drained, except where there was evidence

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of calculi impacted in the common duct. Where the latter was not obstructed the gall-bladder was removed and the duct opened up, cleared, and drained; and in those cases in which the gall-bladder was too friable from inflammatory softening to admit of its being removed without bursting or infection of the surrounding parts it was simply drained.

In only one case was the gall-bladder sewn up after removal of the stone; but here it was suspected that the mass round the cystic duct was malignant and inoperable, and it was desirable to shorten the operation and diminish risks in view of the patient's age (62) and feeble health. Happily, the portion removed showed purely inflammatory tissue.

In only one case was a simple choledochotomy performed, but the stone impacted in the common duct could not be removed until the duodenum too had been opened. The procedure was severe, but recovery followed.

In only one case was it necessary to unite the gall-bladder to the jejunum; and no case could better illustrate the relief derived by this measure where the common duct is blocked by malignant disease.

The group as a whole offers a good picture of what may be done in this particular branch of surgery and its difficulties and dangers. It shows how the latter may be avoided, and above all by not operating *too late*.

September 24th, 1906.

Diagnostic Significance of Decidual Tissue.—As the result of study of a number of cases, Graves concludes that the passage of a decidual membrane in a patient with symptoms of pregnancy and with a mass on one side, together with a history of flowing, is extremely significant of an extra-uterine pregnancy, but that cases do occur in which this seemingly conclusive chain of evidence is not proof of an extra-uterine gestation. An ordinary miscarriage may be preceded by the exfoliation of a part or the whole of the decidua vera. Extreme care should be observed by the pathologist in making a diagnosis, because it may be impossible to differentiate between an exfoliated dysmenorrhœic membrane and the decidua of an extra-uterine pregnancy.—*Journ. A. M. A.*, vol. xlvii, No. 6.

A LECTURE

ON

PYLORIC OBSTRUCTION IN INFANTS.

Delivered at the Hospital for Sick Children, Great Ormond Street, W.C.

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LADIES AND GENTLEMEN,—It is only of quite recent years that the malady which forms the subject of my lecture has attracted general attention, and we must assume that until recently its victims were included in the wide and somewhat heterogeneous group of marasmic infants.

In some instances a malady or group of symptoms has escaped notice until recent times because its recognition was not possible without the aid of minute and elaborate clinical methods which were not at the disposal of our forefathers. Examples of this are afforded by the various so-called blood diseases, the differentiation of which depends largely upon the detection of minute changes and variations in the pathological conditions of the corpuscles of the blood. Bacteriology also, with all its modern developments, plays a very important part in bringing to light new diseases. We are able, by the recognition of the organisms which cause them, to group together morbid conditions which have hitherto been regarded as distinct diseases, and to show, for example, that one organism, the pneumococcus, will produce such diverse maladies as empyema, broncho-pneumonia, croupous pneumonia, meningitis, and others. Setting aside those maladies which owe their late recognition to diagnostic difficulties only to be overcome by the employment of recently discovered methods, there are, even at the present day, groups of symptoms and physical signs which, although they have been under observation for centuries, have only recently, or have not yet, been recognised as constituting definite clinical entities. The progress of clinical medicine has been punctuated by the detection of such conditions by shrewd observers who have realised the significance of obvious associations which their

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forerunners had overlooked. Witness the work of Bright on the association of albuminuria and œdema with disease of the kidneys, and the no less brilliant work of Addison. Many people had been bronzed and had vomited and had had feeble hearts long before Addison recognised that these symptoms occurring in association constituted a definite clinical entity and showed by post-mortem observation that at the bottom of this symptom-complex was caseation of the suprarenal capsules. One might quote many other examples, such as myxœdema, infantile scurvy, and rheumatic nodules. The very first description of rheumatic nodules in medical literature appears to have been as recent as 1868, and they never attracted any wide attention until the year 1875. This group of diseases, viz. those which have failed to obtain recognition until recently, although their features are fairly obvious, illustrate the old parable of "eyes and no eyes," and impress upon us the enormous services rendered by those people who can see things which other people cannot see, and make us feel that, however much we may object on principle to the attachment of the names of individuals to particular diseases, yet the names of Bright, and Addison, and Hodgkin are rightly immortalised in connection with the groups of morbid symptoms of which they first saw the import. To this latter group belongs the disease which forms the subject of the present lecture, and which is characterised by signs and symptoms some of which are so obvious that one marvels that their significance so long escaped recognition.

When a "new" disease is described the question often arises whether it is of recent development. For it may be that sometimes changed conditions of life do predispose to the development of new morbid conditions. It is often a matter of extreme difficulty to ascertain whether a disease such as appendicitis, which is attracting daily increasing attention, is very much on the increase or whether it is merely more often diagnosed. When osteo-arthritis was first described the question was raised whether the older peoples of the earth used to have this disease at all, whether such a malady, characterised by such obvious changes in bones, could have been overlooked if it had existed, and could have escaped notice until the end of the eighteenth century. To that question, fortunately, a very definite answer is forthcoming. Archæology has brought to light the scars of this disease on bones of great antiquity. Again, in the case of the pyloric obstruction of infants it may be stated with confidence that it is no new malady, seeing that its literature can be traced back for some distance. Williamson described a case of this disease in the year 1841, and the erudition of Professor Osler has brought to light a far earlier record in American literature—dating, I think, from 1788. However, it is only within the last six or eight years that the pyloric obstruction of infants has attracted general notice.

There is good reason to believe that the malady is by no means a very rare one. One of my colleagues has in his ward at the present time three perfectly definite cases of the condition, and we are seldom without a case in any medical ward of this hospital.

Although cases of unusual interest naturally tend to gravitate towards a children's hospital, the experience of outside practice bears out the view that it is not a rare disease, and that the more widely it is looked for and known the more frequently are the cases recognised. The examples which I have met with have been without exception in male infants, and others have had like experience, pointing to a special liability of the male sex, but on referring back to the recorded cases in the literature one finds that the supposed predominance among males is not so great as one's own experience would indicate.

Vomiting is the first symptom of the disease which attracts attention, and vomiting of a peculiar kind. It is hardly necessary to point out that vomiting is of extremely common occurrence in infancy, and that the symptom has very different significance in different cases. In the first place, almost every infant throws up, at some period of its career, portions of its food, either at the end of each feed or at the end of some feeds. When the stomach is full a part of the contents tends to overflow upwards, and to such overflow vomiting little importance attaches. There are, it need hardly be said, many other kinds of vomiting met with in infants, such as that due to gastro-enteritis, that due to improper feeding, the mere irritation of the alimentary canal by unsuitable food at too early an age. Again, the cerebral vomiting of infants calls for mention, such as constitutes a prominent feature in most cases of post-basic meningitis. This is apt to occur on groups of days:

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that is to say, for a few days there will be frequent vomiting, and for a further period there will be none at all, or hardly any. It is obvious that the mere occurrence of vomiting affords but scanty evidence of pyloric obstruction, and that the diagnosis of this condition must in no case be based upon this symptom alone. It is true that the vomiting of pyloric obstruction is of a peculiar kind, and its peculiarity manifests itself in several ways. In the first place, it is not incessant and does not occur after each successive feed, but as a rule only once or twice in the twenty-four hours. The vomiting is also peculiar in regard to the quantity brought up. It is a very noticeable fact that in cases of pyloric obstruction the amount vomited is apt to be considerably greater than the amount of the last meal. Obviously the child has stored up in its stomach the last meal, and sometimes several successive meals. There is difficulty in emptying the stomach onwards, and the vomiting bears witness to the accumulation which has taken place in a stomach dilated to much beyond its ordinary size. A third peculiarity of the vomiting of pyloric obstruction in infants is the force with which the food is expelled. The stomach is emptied rather suddenly and with considerable violence, a fact which points to abundant muscular power in the walls of the dilated viscus. So violent may be the ejection that the stomach contents may be expelled to a distance of several feet from the patient. Such vomiting is strongly suggestive of the presence of pyloric obstruction. It does not usually begin in the first days of extra-uterine life, and the commonest time for its commencement is from the third to the fifth week after birth. After the vomiting has commenced the child soon begins to show the effects of the pyloric constriction. Seeing that very ittle passes on into the intestines, the stools are naturally scanty in volume. Constipation is the rule because of the drain of liquid, due to the copious vomiting which occurs. Thus the characters of the stools contribute to the recognition of the condition in question. The vomit of a child fed on milk often contains, as would naturally be expected, large quantities of curd, and one feature which may be noticed is that it never contains bile. Some writers lay stress on this absence of bile as a special feature of the condition, and as showing that, owing to the pyloric obstruction, the bile does not make its way from the duodenum into the stomach and is not vomited, but I do not think that this feature is in any way peculiar to this particular variety of infantile vomiting.

Of very great diagnostic value, when taken in connection with such vomiting as has been described, is the gastric peristalsis which forms one of the most prominent features of these cases. The peristaltic waves, which may be so salient and conspicuous as to be easily shown in a photograph, traverse the elongated and dilated stomach from left to right, in slow and deliberate sequence. Frequently three distinct waves can be seen at the same time, one emerging from beneath the ribs on the left side, one just about the centre of the dilated stomach, and one passing away towards its pyloric end. The fact that the peristalsis proceeds from left to right serves at once to distinguish it from a peristalsis in the transverse colon, which, under ordinary circumstances, will necessarily be from right to left. Dr. Still, in one of his papers on the subject, mentions that he has seen a peristalsis from left to right in the upper limb of a dilated sigmoid flexure. Naturally such peristalsis would take this direction, and it is worth noticing that so good an observer as Dr. Still has actually known difficulty to arise from the possibility of mistaking peristalsis in a dilated sigmoid for that in the stomach. As a rule the waves mark out clearly the position and lie of the stomach. It is noticed sometimes that the stomach lies peculiarly low, and gives the impression of a tubular rather than of a widely dilated viscus. Without obvious gastric peristalsis the diagnosis of this variety of pyloric obstruction cannot be made with any approach to certainty, and this symptom yields in importance to no other sign of the disease under discussion.

I have here a photograph (see Fig. 1) which shows one of the peristaltic waves. It appears as an evident tumour of the size of half a Tangerine orange tracking across the abdomen and in its course it maps out the form and position of the stomach.

The third cardinal physical sign of the condition is the presence of a palpable pyloric tumour. There are cases which are undoubtedly examples of the disease in question, in which for days at a time no pyloric tumour can be felt in the course of an ordinary cursory examination. However, in

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almost every case which can be safely and definitely diagnosed as an example of pyloric obstruction the pyloric tumour is felt, provided that it is patiently sought for day after day. Sometimes it can easily be detected for days together, and then for a further period it may entirely elude detection. In cases which tend towards recovery the permanent disappearance of the pyloric tumour may be ranked as one of the favourable signs, just as it is certainly the case that the diminution and less obvious character of the peristalsis must be regarded as a favourable sign. Undoubtedly the pylorus becomes more palpable when the peristalsis is at its height, a fact which suggests

FIG 1.

that its palpability is greatly increased by pyloric spasm, such as may be supposed to accompany the traversing of the stomach by very conspicuous peristaltic waves.

These, then, are the cardinal symptoms of the disease: copious vomiting of a peculiarly forcible character, and at somewhat long intervals, obvious left to right peristalsis in the dilated and elongated stomach, and a palpable pyloric tumour. If these three signs are present the diagnosis may be taken to be established beyond doubt. On the other hand, there are cases in which one or more of these signs are absent, but in which the existence of pyloric stenosis is highly probable. In such cases the probability warrants the adoption of appropriate treatment, for therapeutics cannot always afford to wait upon scientific certainty, but as bases of statistics they should be left out of account.

Rapid and conspicuous wasting is an inevitable result of any pronounced degree of pyloric stenosis. It is obvious that if an infant stores in its stomach a large portion of the food which it takes and at intervals rejects it, whilst at the same time only a comparatively small stream of nutritive material is passing on through the pylorus into the intestine, the nutrition of the child must seriously suffer. An infant which vomits only once a day will not exhibit the extreme degree of wasting here seen, provided that the vomited material only represents a single meal.

Numerous post-mortem examinations have led to the acquisition of a fairly complete knowledge of the morbid anatomy of the disease. I have before me some specimens from cases occurring in this hospital which illustrate the points which call for mention. The main feature is a hyperplasia or hypertrophy of the circular muscle of the sphincter of the pylorus. The mucous membrane and submucous tissue is apt to be thickened, and in some of the earlier descriptions considerable stress is laid upon such increase of the submucous tissue, for there can be no doubt that the infolding of the mucous membrane does contribute largely to the obstruction which the condition produces, and often renders the pyloric orifice very narrow indeed. One fold of the mucous membrane is apt to be specially conspicuous. It corresponds as a rule with the lesser or greater curvature of the stomach, and has been compared to the crista galli. However, the hypertrophy or hyperplasia is mainly of the muscular fibres of the pylorus, and of the circular fibres rather than of the longitudinal.

In approaching the study of the pathogeny of the condition, we are confronted with an as yet unsolved problem, and one of very considerable difficulty. It is held by not a few of those who have devoted special study to the subject that the primary evil is a malformation, a congenital hyperplasia, of the muscular coat of the pylorus; others, again, maintain that the primary trouble is of the nature of a spasm, and that to this spasm the hypertrophy is secondary. Considerable obstacles present themselves to the unqualified acceptance of either of these alternative views. In

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the first place, it is not easy to quote any other malformation at all comparable to a congenital hyperplasia of the pyloric muscle; but in favour of the malformation theory is the undoubted fact that pyloric lesions such as we are discussing have been seen in the unborn fœtus, a fact which makes it necessary to grant that the lesion may develop in fœtal life. It is true that in the cases which come under clinical observation the onset of the symptoms at the third, fourth, or fifth week of life, and not immediately after birth, suggests that in most instances the commencement of the trouble is post-natal, but this does not detract from the importance of the fact that an exactly similar lesion may exist before the child is born. Dr. John Thomson, who inclines strongly to the view that spasm plays a very important part in the malady, suggests that the fœtus *in utero* continually swallows amniotic fluid which passes along the alimentary canal, and he suggests that spasm may begin *in utero*, and that the hypertrophy may result therefrom. On the other hand, those who reject the spasmodic hypothesis allege that the rapidity with which the great hyperplasia or hypertrophy is produced is incompatible with its being merely the result of excessive muscular activity; and there is considerable force in the argument which they put forward, that such hypertrophy of unstriped muscular structures, such as the sphincter ani, is unknown as the result of spasm. On the other hand, the clinical features of the disease, and especially the more ready palpability of the pyloric tumour when the stomach is being traversed by peristaltic waves, suggests that spasm of the pylorus has, at any rate, a conspicuous share in producing the obstruction of the orifice.

The prognosis in the pyloric obstruction of infants is, undoubtedly, very unfavourable, and it seems probable that in pronounced cases which are not recognised and are not treated a fatal ending is almost inevitable. It is equally probable that there are all degrees of this condition, that the cases which show flagrantly all the characteristic physical signs are merely the extreme examples, and that minor degrees often occur and are often recovered from, even apart from any definite course of treatment. One thing is certain, that apart from surgical means—and cases surgically treated may in the present connection be put on one side—recovery is sometimes brought about by careful nursing and careful feeding. The malady is not necessarily fatal apart from surgical treatment—a point of extreme importance in deciding the question whether or no surgical interference should be resorted to in any particular case. If it were a condition which was necessarily fatal if unrelieved by surgery, and if it could be shown, as it can be shown, that when treated surgically a considerable proportion of the patients recovered, obviously we should have no alternative but to urge surgical interference in every case in which the diagnosis was beyond question, and in which the condition of the child seemed to render an operation possible or warrantable.

Let me now demonstrate to you two cases. This is a child which has been under treatment in Alexandra ward for some time. The story is that three weeks before admission the child had constipation and vomiting and that the motions resembled marbles. There was no blood or slime in them. The infant had all the characteristic symptoms of pyloric stenosis. The photograph which I passed round (Fig. 1), showing the peristaltic wave, was taken from this child. Since that date the child has been treated in ways which I will describe to you presently. The weight, which is perhaps the best criterion of progress, is recorded in this chart, and you will notice some interesting points in connection with it. You will see that the weight of the child has repeatedly risen to 12 lb., and has repeatedly decreased again. It appeared as if this weight represented a kind of maximum in view of the size of the pylorus, and that the metabolism was balanced at that point. But a few days ago the 12 lb. weight limit was exceeded and is now largely exceeded, so that we have great hopes that we are on the high road to improvement.

You will notice, in the first place, the emaciation of the child. At present I cannot demonstrate to you the peristalsis, because the room is too cold for the exposure of so delicate an infant. In this case the pylorus has been repeatedly felt, and for days together it could be felt with great confidence and certainty. Recently it has not been so well felt, emphasising the fact to which I have already referred that palpability of the pylorus and diminished peristalsis are among the more important signs of improvement. The second child which I would bring to your notice offers a most con-

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spicuous contrast to the last. Although now rosy and robust, with all the signs of perfect health, and, if anything, rather too fat, this patient exhibited all the cardinal symptoms of pyloric stenosis, and, at a corresponding age, was quite as wizened and emaciated as the infant whom you saw just now. The vomiting was copious and forcible, the gastric peristalsis was as conspicuous as in any case that I have seen, and the pyloric tumour was readily palpable. The child was in the hospital from February 16th to June 30th, 1905, and his case was fully described by our former house physician, Mr. Blaxland, in the 'Lancet' of last September. He has now reached such an age that he offers an excellent example of recovery apart from surgical treatment. This raises the very important question: In what does such recovery consist? Can we assume that the pyloric hypertrophy or hyperplasia disappears? Looking at the child, one would believe that it is perfectly well, that whatever the mischief was it is now altogether at an end. However, there have been a few cases recorded in which, within a few months of apparent recovery or of the disappearance of the symptoms, the child has succumbed to intercurrent disorders of various kinds, such as broncho-pneumonia, diarrhœa, etc. One of these was a case under the care of Dr. Batten, in this hospital. In such cases the pyloric hyperplasia has been found to be as marked as in cases of death directly due to the stenosis, and the stomach has been found still to be dilated and hypertrophied. These observations suggest that what appears to be recovery from this disease is merely the development of such hypertrophy of the stomach as a whole as enables it to overcome the effect of the pyloric stenosis—in other words, that there is no true recovery, but a compensatory hypertrophy comparable to that of the left ventricle of the heart, which may put into abeyance the symptoms of aortic regurgitation.

If it be the case that the recovery of the second child which I have shown to you to-day was of this nature, the very satisfactory general condition and the absence of any sign of impaired nutrition are at least remarkable. If the obstruction is still present, and is only overcome by the power of the walls of the hypertrophied stomach, it is curious that there should be such a complete cessation of vomiting as in this case, and that the hypertrophied stomach should no longer be the seat of visible peristalsis, bearing witness to the difficulty encountered in propelling the gastric contents through the narrowed orifice.

The further question of what eventually happens in cases of recovery without operation is one to which no answer can as yet be given. It is only in quite recent years that these cases have been diagnosed in any numbers, and none have yet been watched up to the later stages of childhood. There undoubtedly are cases of simple non-malignant stricture of the pylorus occurring in later life, and it has been suggested that such strictures may be legacies of the slighter degrees of the infantile malady, which have not proved fatal in babyhood, but have led to trouble later on. Some six months ago there was in my ward a child of eight or nine years, with an obvious dilatation of the stomach which was found to be due to simple pyloric hypertrophy, for the relief of which gastro-enterostomy was performed by my colleague, Mr. Arbuthnot Lane. It is possible that in that case the pyloric hypertrophy may have had its origin in infancy or in intra-uterine life, although the symptoms were of comparatively late development.

The remainder of the time at my disposal must be devoted to questions of treatment, and the treatment of the pyloric obstruction of infants may be considered under the several headings of diet, effects of drugs, gastric lavage, and operation.

The diet suitable to any particular case can only be determined by experiment; no definite rule can be laid down, for the nature and quantity of the food given will depend upon observation from day to day and from week to week. The weight of the patient and its fluctuations, the character of the vomit, and the frequency of the vomit, all serve as guiding signs. In the cases of the infants whom you have seen this afternoon peptonised milk, humanised milk, asses' milk, whey, white wine whey, and plasmon-water have all been employed in turn. Cream has been added to the diet at times. In the second case as progress was made towards recovery the diet gradually approximated to that of a normal child of corresponding age. Food should be given in small quantities and at short intervals.

Drugs are apparently of little value. Opium has been tried, as also hyoscyamus, belladonna, and cocaine in minute doses, but it appears to me that if we are to save a child without operation it must

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be by very careful nursing and dieting, by washing out by the stomach, and by watching the case carefully from day to day and regulating the treatment according to circumstances which arise from time to time.

Lavage of the stomach is a very valuable method of treatment in many cases of pyloric stenosis, and its employment has been strongly advocated by my colleague, Dr. Still. If spasm plays an important part in the disease we can well imagine that lavage may be of much service, for by washing out the accumulated curds in the stomach from time to time we give the stomach a fresh chance once or twice a day. The treatment should be carried out with circumspection, and may have to be discontinued from time to time. Both the cases which you saw have been treated by washing out the stomach. Sometimes the lavage has been stopped for a time; sometimes the stomach has been washed out once a day, sometimes twice. As the second child progressed towards recovery the lavage was carried out less and less frequently, and was eventually wholly discontinued.

The treatment of these cases calls for most careful watching and first-class nursing, and it is mainly due to the skill and care of the sister and nurses of the ward that the fat baby which I showed to you recovered at all. The slightest relaxation of care on their part would have led to its death. It will readily be imagined that the adequate treatment of such cases in private practice imposes a very severe strain alike upon the nurses and upon the practitioner in attendance.

Although such treatment as has been described is in some cases attended by very satisfactory and encouraging results, it must be confessed that the disappointments far outnumber the successes. Even in the cases which do best the issue hangs long in the balance and at times one feels that the scales are trembling. The reserve of strength left to the little patient is very small and it takes little to turn the scale in the direction of a fatal ending, and even when after a long and anxious period the infant seems to have started on the high-road to recovery, some intercurrent disorder, usually broncho-pneumonia, renders all one's efforts vain.

Thus one is often placed on the horns of a dilemma. If medical treatment is given a fair trial the stage at which a surgical operation offers a fair prospect of success will probably be passed. If the surgeon is called in when the signs indicate that medical treatment will prove unavailing, the condition of the infant will probably lead him to decide against operation. If again the case once more enters upon the upward grade one hopes that after all no operation will be necessary. It is, for this reason, incumbent upon us to endeavour to decide once and for all whether the aid of the surgeon shall be invoked in any particular case, at an early stage, whilst the condition of the patient is still as good as possible, for if systematic treatment by lavage be once embarked upon it is probable that before it can be fairly tested the opportunity of successful surgical interference will have passed. How difficult such a decision is only those who have had occasion to treat such cases can adequately realise.

If an operation is resorted to early in all cases, it is certain that some cases will be operated upon in which recovery was possible without surgical measures, and an abdominal operation on a child of five or six weeks of age is a serious undertaking, not lightly to be entered upon. On the other hand, there are no means by which one may recognise cases which offer a reasonable chance of recovery without operation, at any rate among those in which the cardinal symptoms are so well marked that the diagnosis is sufficiently well established to justify an operation.

I do not propose to say much as to the purely surgical question of the nature of the operation which should be preferred. Pylorectomy has, I believe, been only once performed in such a case, and is not likely to be repeated.

Loreta's operation has been performed in some cases, and it has been well thought of by some surgeons. Mr. Dent, who has, in co-operation with Dr. Cautley, performed a number of operations in cases of this kind, leans to pyloroplasty as the most satisfactory operation. It is comparatively quickly done, there is not much exposure of the abdominal contents, and he believes it to give the most satisfactory results. Other surgeons advocate gastro-enterostomy. It is curious that if one refers to the literature of the subject one finds that abdominal surgeons of very great authority, who have, however, had comparatively small experience with this particular class of case, tend to advocate gastro-enterostomy as undoubtedly the operation to which preference should be given. On the

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other hand, those surgeons who have had most special experience of the pyloric obstruction of infants prefer pyloroplasty or dilatation of the pylorus to gastro-enterostomy in the treatment of this particular condition.

I am afraid that you will consider that I have spoken with a very uncertain voice on the question of the desirability of operation in the disease under discussion. I have spoken vaguely because my mind is not yet fully made up. I think that if experience shows that any considerable number of such infants can be saved without operation, then in view of the seriousness of abdominal operations in children in the first months of life one is bound to do all one can so to save them, whereas if, as I said before, it had been shown that without operation the condition was necessarily fatal, no one would more earnestly advocate surgical interference as a routine measure. I have endeavoured to show you how many disappointments one is apt to meet with in cases medically treated, in spite of an occasional brilliant success; and if the perfection of the operative methods should give results so favourable that they may be embarked upon with a comparatively light heart, the shortening of the critical period, with its many attendant risks, will supply a very strong argument in their favour. However, even in some cases in which operations have been performed the after-feeding of the patients has been attended with great difficulties, and the recovery from the operation itself does not always mark the end of the vicissitudes of the cases.

I trust that this brief and very imperfect account of a malady which, although it is attracting considerable interest, is not yet so familar to all as it might be, may have the effect of stimulating your interest, and of putting you in possession of such main facts concerning it as will enable you to diagnose such cases when you come across them.

September 24th, 1906.

RIESE has removed the seminal vesicles in seven cases, operating through a transverse incision in the perineum. In five tuberculous cases the patients were restored to health although there were tuberculous lesions in the lungs and bacilli in the sputum.—*Journ. A. M. A.*, vol xlvii, No. 6.

RADIOTHERAPY IN SKIN DISEASE. By DR. J. BELOT. Authorised Translation from the Second French Edition by W. DEANE BUTCHER, M.R.C.S., Surgeon to the London Skin Hospital. Published by Rebman, Limited.

The growth of radiotherapy, born but yesterday, is so phenomenal that it has already made enormous strides, and to-day it may be said to dominate the therapeutics of dermatology. This work of Dr. Belot's is already well and favourably known on the Continent, where it has reached a second edition. We welcome this excellent translation by Mr. Deane Butcher, which has made a most important addition to the English literature of the subject. The work is divided into three parts. The first part contains a historical review of Röntgen's discovery, which is followed by a brief but lucid description of the various forms of apparatus used for the production and measurement of the Röntgen rays. An interesting review of the properties of the Röntgen rays and the hypotheses as to their nature concludes this section. The second part opens with a study of the nature of the radiations from an X-ray tube. After having shown that it is the X rays alone which are therapeutically active, Dr. Belot discusses the principal factors which govern their application. The third part, purely clinical, appeals most to the medical practitioner. It deals systematically with the many and various dermatoses in which radiotherapy has been employed. It is interesting to examine the results of the application of X rays to cutaneous epitheliomata. Many cases are recorded in detail with photographs taken before and after treatment. "It will be seen," says Dr. Belot, "that the percentage of successes is very satisfactory. In most cases the patients were completely cured by a month's treatment. As regards the permanency of the cure, the statistics which have appeared up to the present time are most encouraging. But are we yet justified in forming a very optimistic opinion on the value of radiotherapy? We have not had sufficient length of experience to say. When the patients apparently cured have remained so for four or five years it will be time enough to answer this question. Meantime we do not assert that cancer of the skin can be permanently cured by this method. What we do affirm is that radiotherapy temporarily removes the objective symptoms of nearly all cutaneous neoplasms, that the cicatrices are most satisfactory, and that the chances of recurrence are less than in any other mode of procedure." The utility of this volume is unfortunately diminished by the absence of an index.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE ON SOME FORMS OF SURGICAL TUBERCULOSIS IN CHILDREN.

Delivered at University College Hospital.

By RAYMOND JOHNSON, B.S., F.R.C.S.,
Surgeon to the Hospital and to the Victoria Hospital for Children.

GENTLEMEN,—I need hardly say that it is with only a very few aspects of such a wide subject as the surgical forms of tuberculosis in children that we shall be concerned this afternoon. And first of all let me say a few words with regard to the disease in quite young children—infants, perhaps only a few months old. Although such affections as tuberculous disease of the spine and large joints are occasionally seen even at this early age, they are not at all common.

Among the most common sites of tuberculous disease in early infancy is the skin, and the lesions are very often multiple. They begin as a small, hard nodule in the skin, which, as it enlarges softens, while the skin over it becomes thin and discoloured. A small chronic abscess is, indeed, formed, which if untreated will probably discharge and sometimes quickly heal, although it may result in an intractable tuberculous ulcer. The most that is required in the treatment of one of these softened tuberculous nodules is to puncture it and scrape it with a small, sharp spoon, when it will usually quickly heal. I must remind you, however, as a case to be quoted presently will show, that these tuberculous skin-nodules may sometimes rapidly shrink spontaneously, even after having obviously softened, and disappear, leaving behind only a small cicatrix.

Another form of surgical tuberculosis not uncommon at a very early age is tuberculous dactylitis, and it is often seen in association

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with the skin-deposits just mentioned. You are all familiar with this common affection, which is much more frequently seen in the hand than in the foot. The bones usually affected are the metacarpals and first phalanges, especially the latter. A painless enlargement of the bone is accidentally discovered, and, although in some cases the swelling subsides without suppuration, chronic abscess is the usual result, the abscess most commonly appearing on the dorsum of the hand or the lateral aspect of the finger. The disease begins as a rule in the interior of the bone, as a tuberculous osteo-myelitis, and, fortunately, in a large majority of cases the joints escape. I may remind you that the four inner metacarpal bones possess an epiphysis at their distal extremities, whilst the metacarpal bone of the thumb and all the five proximal phalanges have an epiphysis at their proximal extremities. Judging from the analogy of the long bones of the limbs, it is probable that the actual starting-point of the disease is in the immediate neighbourhood of these epiphyses.

Operation for tuberculous dactylitis is only indicated when an abscess has formed and should not be carried out too vigorously lest the neighbouring joints and tendon-sheaths be damaged. A small incision made into the abscess will generally lead directly into the interior of the expanded bone, from which the tuberculous tissue, and often a small sequestrum, can be carefully scraped with a small spoon. A little undiluted carbolic acid is then applied and the hand enclosed in a gauze dressing, which in a young child will also act sufficiently as a splint. The dressing may often be left unchanged for two or three weeks, when in a fair proportion of cases the wound will be found healed.

It is interesting to observe the after-effects of this disease in adult life; the affected fingers generally show marked arrest of growth, but often retain their movements almost unimpaired. Amputation for tuberculous dactylitis should only be resorted to as a last resource, and is rarely necessary unless one of the joints has become disorganised. I have never seen a case of tuberculous dactylitis affecting the second phalanx primarily, but in two instances have seen the disease in the terminal phalanx. In one such case, a boy of ten years, who had also a tuberculous gland in the neck, the disease of the last phalanx had the remarkable effect of causing an enlargement of the whole end of the thumb, including the nail, so that it was as large as that of a man. I saw this boy two years later, and the whole thumb, with the nail, had returned to its normal size.

I have often observed tuberculous disease of the supra-epitrochlear lymphatic gland in association with tuberculous dactylitis, and have seen the same gland involved in a case of tuberculous teno-synovitis at the wrist. The supra-epitrochlear gland lies in front of the internal intermuscular septum, a varying distance above the internal condyle of the humerus. It is, I believe, very rarely the seat of secondary abscess in the very numerous acute suppurative conditions about the hand and fingers. In these cases the axillary glands are those usually affected. I have no explanation to offer for the difference in the two classes of cases.

A form of dactylitis is described as an occasional manifestation of congenital syphilis. If such a condition really exists it must be extremely rare, and the possibility of tuberculous dactylitis occurring in a child the subject of congenital syphilis must be borne in mind.

In illustration of some of the points above mentioned I may quote the following cases: A male child, æt. 9 months, was brought to the Victoria Hospital for Children, with tuberculous deposits in the skin of the scalp, cheek, buttocks, thigh, leg, forearm, and hand. The deposits varied in size, and over some which had softened the skin was thin and red. There was a mass of suppurating glands in the neck. The child was thin and delicate-looking, with flushed cheeks and dry, loose skin. The cutaneous abscesses on the scalp and cheek, and the glandular abscess in the neck, were opened and scraped. Two months and a half later it was noted that the child's condition was much better; all the skin-deposits without any local treatment were shrinking and the redness of the skin over them had disappeared. At the age of nineteen months the situation of the deposits could only be recognised by the slight scars which they had left. Other tuberculous lesions had, however, made their appearance; there was a softened gland above the left clavicle, and a large abscess over the upper end of the right tibia, which required treatment. Both hands had been

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noticed to be swollen for a fortnight, and looked at first sight as if they were œdematous. Examination, however, showed that the swelling was due to enlargement of the first, second, and fifth metacarpal bones of the right hand, and the second, third, and fourth of the left. The child had the advantage of being well fed and cared for, and when I last saw him, at the age of two years and seven months, he appeared to be in robust health, with the exception of slight phlyctenular conjunctivitis and eczema of the cheek. All the tuberculous lesions had become quiescent, and the enlargement of the metacarpal bones had entirely subsided.

A male child, whose mother's sister and father had died of consumption, was brought up on cow's milk and barley-water. At twelve months it had chronic abscesses about the left ankle, in the metacarpal region of both hands, in both forearms, and in the glands of the neck. Numerous operations were performed, and at the age of two years and a half the child seemed in good health, and all the tuberculous deposits had firmly healed except that in the ankle. Five months later this also had healed, but an abscess in the right supra-epitrochlear gland had recently discharged spontaneously and was healing.

Such stories might be often repeated, showing a widely spread tuberculous infection, followed by an apparent restoration to perfect health. The ultimate result is, however, less satisfactory, and the comparative infrequency with which evidences of these multiple tuberculous lesions are met with in adults can only be explained in one way. The tuberculous infant, although patched up for the time, frequently succumbs to some other manifestation of the disease before reaching adult life.

The source of the infection in these cases has been the subject of much investigation. When we see a child with perhaps dozens of scattered superfluous tuberculous lesions arising almost simultaneously in various parts of the body, we can hardly regard any one of them as primary, but may safely assume that they are all secondary to some hidden caseous focus, probably in the glandular system of the chest or abdomen.

Three facts have struck me in connection with these tuberculous infants, as they present themselves in the Out-patients' Department: (1) the strong influence of heredity, the child's father in quite a number of cases having recently died of tuberculosis; (2) the rapidity with which multiple tuberculous lesions, especially in the skin, may manifest themselves; (3) the striking way in which the infant will get the better of the disease, recovering almost as if from an acute illness, the local lesions undergoing involution, and the general nutrition of the child showing a corresponding improvement.

Another tuberculous affection, the frequency of which is difficult to estimate in young children, is tuberculous disease of the middle ear and mastoid process. It is probably much more common than is usually believed, because it may not present clinical features sufficiently characteristic to distinguish it from the non-tuberculous affection.

Tuberculous disease of the middle ear may be unilateral or bilateral, and in its course behaves like a tuberculous bone-abscess in any other situation. Thus, a very exuberant formation of granulation-tissue occurs in the tympanum, and this may extend through the perforated membrane so as to form a granulation polypus in the external meatus, which will rapidly recur if merely scraped away. The cavity of the tympanum becomes rapidly enlarged by caries of its wall, and the disease spreads into the mastoid process. In the latter the bone-destruction is often strikingly rapid, and an abscess quickly forms behind the auricle. On opening this the widely-spread nature of the disease is at once apparent, and often a large, spongy sequestrum is readily removed by scraping.

The disease of the bone often involves the aqueduct of Fallopius, and facial paralysis is the result. Indeed, I may remind you that the occurrence of facial paralysis in a child suffering from chronic otorrhœa should always suggest that you are dealing with a case of tuberculous disease. Another frequent result is the extension of the disease to the lymphatic glands around the ear.

The treatment of a case of tuberculous disease of the middle ear should be carried out in a thorough manner by freely opening up the mastoid antrum and tympanum, and removing the tuberculous granulation-tissue and carious bone by careful curetting. It is probably best to leave the posterior wound widely open, and plug the cavity from it rather than by way of the meatus, which in a young child can hardly be enlarged sufficiently to gain satisfactory access to the cavity.

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I could tell you of many cases of mastoid disease in young children, in which one or other of the signs I have mentioned indicated their tuberculous nature. In one instance a boy of two years had simultaneous disease of both middle ears and mastoid processes, for which operation was performed. On the right side healing rapidly followed, but on the left side a mastoid sinus persisted. A year later evidences of caries of the spine showed themselves, and a double Thomas's splint was applied. A psoas abscess formed on the left side, and was successfully treated by incision and flushing. In the following year a second operation was performed on the left mastoid, which then also healed, and now the boy is five years old and seems in every way to be doing well.

I have at the present time a child under my care who last year had mastoid disease and facial paralysis, and now has tuberculous dactylitis. The facial paralysis was recovered from after the mastoid operation.

The early diagnosis of tuberculous disease of the spinal column in children is often a matter of the greatest difficulty, but of the most grave importance. That the difficulty is great is sufficiently indicated by remembering what a variety of clinical forms the disease may assume at the outset. One child is accidentally noticed to have a spinal deformity, another complains of "stomach-ache," in another the abdominal tumour caused by a spinal abscess is the first indication of the disease, and in yet another the gradual onset of paralysis of the lower limbs may be observed before any clear evidence of the disease of the spine can be recognised.

I am not concerned with the general aspects of this important subject, but I want to call your attention to two matters only in connection with it. In the first place I want to remind you that in an infant difficulty is sometimes encountered in distinguishing a curve of the spine due to rickets from a curve due to tuberculous disease. I do not think the difficulty in this connection is often very great, and of course the presence or absence of other evidences of rickets or tuberculosis will help. The deformity in rickets is usually a kyphosis of considerable extent and sweeping curve, involving the lumbar and dorsal regions, whereas in the curvature of tuberculous disease the extent of the curve is much more limited, and it is usually so abrupt that it is really angular and presents a distinct apex. Moreover, in the rickety spine the degree of mobility is much greater than in the diseased part of the tuberculous spine. This can be tested by laying the child on its face and lifting up the pelvis by the feet.

I want more particularly, however, to refer to the difficulties in the early diagnosis of tuberculous disease of the cervical spine in children. Painful stiffness of the neck is usually the earliest symptom, but fortunately it is only in a minority of the children who have this symptom that it is due to spinal disease. The position assumed by the head may exactly imitate other forms of wry-neck of muscular origin, such as that which may occur as the result of some painful condition in the neck, particularly inflamed glands, or a form of wry-neck sometimes occurring as a manifestation of rheumatism. I know of no certain means of diagnosis in such cases. It is unlikely that examination of the line of the spinous processes will help, and at this early stage it is still more unlikely that any evidence of retropharyngeal abscess will be detected by passing a finger to the back of the throat. Radiographic examination can hardly give positive evidence, and a negative result in such cases is useless as a help in the diagnosis. What, then, is the right line to adopt? Give the child an anæsthetic to produce complete muscular relaxation, and you will probably find that all rigidity disappears and the head may be very gently rotated and moved in other directions quite freely. This, of course, does not prove that there is no spinal disease any more than free mobility of a stiff hip under an anæsthetic proves that the hip-joint is not diseased. In both cases the fixation is due to involuntary muscular contraction, and may entirely disappear during general anæsthesia.

The head is gently put into a straight position, and the child laid flat on a mattress with the head steadied by sand-bags. At the end of a fortnight the sand-bags may be removed and the child still kept lying flat on the back, but allowed to move its head from side to side as it seems inclined. If no stiffness or pain results the child may be allowed to sit up a week later, and the course of events will soon show if the symptoms were due to some trivial muscular condition or to more serious disease of the spine.

The treatment is, indeed, carried out on pre-

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cisely similar lines to those adopted in cases of recently developed limitation of movement in a large joint, such as the hip, when the question Is it early tuberculous disease or not? cannot at once be answered. Complete rest should be enforced for at least a fortnight, and if limitation of movement still persists, or returns when more freedom is again allowed, the existence of joint-disease (for disease of the spine may for practical purposes be regarded as joint-disease) should be suspected and suitable treatment adopted. As regards suitable treatment for early cervical caries in a young child, I should strongly advocate a double Thomas's splint with a suitable padded head support. In an older child rest on the back is essential, with the head fixed by sand-bags; or, if pain is not thus relieved, with a light weight extension attached to the head. As far as possible this part of the treatment should be carried out on a suitable couch, so that the child may be got into the open air.

Before concluding I want to bring before you a few cases illustrating a somewhat uncommon but important form of tuberculous disease, viz. that of the bursæ over the great trochanter. I should not refer to this matter merely from the point of view of pathological interest, but because the subject has a special clinical interest, on account of the fact that the disease of the bursæ is one of the causes of a chronic abscess simulating the common variety of abscess resulting from disease of the hip.

In Quain's 'Anatomy' three bursæ are described over the great trochanter—(1) a large multilocular bursa, or two or three smaller ones, between the fascial insertion of the gluteus maximus and the great trochanter, and another between it and the upper part of the vastus externus; (2) a small bursa between the gluteus medius and the upper part of the trochanter; and (3) a bursa between the tendon of the gluteus minimus and the tubercle of the femur.

I once had an opportunity of performing a post-mortem examination upon a child in whom there was a tuberculous abscess of the large bursa beneath the insertion of the gluteus maximus. The boy died in this hospital from a tuberculous tumour of the cerebellum. Two months before his admission the right hip had been bruised by a fall, and soon afterwards a swelling was noticed over the trochanter. On admission, the boy presented characteristic symptoms of a cerebellar tumour. There was also a fluctuating swelling over the right great trochanter, but there were no other evidences of disease of the hip-joint. Death occurred in coma on the ninth day. The abscess over the hip led down to a caseous mass on the outer aspect of the trochanter beneath the gluteus maximus. The periosteum was intact, and it may be taken as certain that the disease had begun in the bursa. It is important to note, as you might have anticipated, that there were caseous and calcareous bronchial glands, and some old and recent tubercles in both lungs.

I should like, also, to refer very briefly to three cases of this kind upon which I have operated during the last year or two.

Case 1.—A girl, æt. 10 years, complained of pain in the left hip and knee. This was early in 1903, and at the following Christmas a swelling was noticed over the outer aspect of the hip. In April, 1904, the girl was admitted into the Victoria Hospital with a large chronic abscess over the great trochanter. This was opened and found to lead down to a caseous mass on the trochanter, which was dissected out and lay superficially to the periosteum. The incision was closed, but a small sinus persisted for nearly a year and then healed. This was a year ago, and I have recently seen the girl, with no signs of the disease except a firmly healed and depressed scar.

Case 2.—A girl, æt. 6 years, developed symptoms of dorsi-lumbar caries in April, 1903. In February, 1904, she first came under my care with well-marked angular curvature and a right psoas abscess. The psoas abscess was opened, flushed, and closed, and the child sent to Broadstairs on a double Thomas's splint. The psoas abscess rapidly refilled and had to be drained, but soon healed. An abscess now formed over the left hip, and presented, on being opened, a condition exactly similar to that in the last case, and was clearly not an extension of a spinal abscess. The abscess was drained, and after a few months had healed. This was in December, 1904. I saw the patient again in November, 1905, and found everything satisfactory except that she had phlyctenular conjunctivitis.

Case 3.—A girl, æt. 11 years, was treated for a chronic abscess over the great trochanter early in 1905. She had no signs of hip disease, and at the operation the disease was found to be superficial to the periosteum of the great trochanter. In a few months the small sinus healed and when I recently

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saw the girl, about a year after the operation, the scar was firm and there were no evidences of disease.

At the present day extensive and formal operations on the tuberculous joints of young children are as far as possible avoided. In dealing with a tuberculous abscess in the neighbourhood of one of the large joints it is very necessary to proceed cautiously and not jump to the conclusion that the joint itself is necessarily involved. The abscess should be freely opened and cleaned of its contents. The edges of the incision being now well retracted and the cavity illuminated, the track which leads more deeply to the actual disease can usually be found. By enlarging this track the disease itself is at last reached. This may be in the joint itself, but not infrequently it is situated in one of the bones or even in some neighbouring bursa or tendon-sheath. The operation may thus be the means of preventing infection of the still healthy joint, instead of seriously increasing the risk of this occurrence.

October 1st, 1906.

"State of Morbid Imminence."—By this term Londe means a condition in which the organism is depressed from some cause, ready to succumb to the first infection or other malady for which opportunity offers. The diseases apparently most sudden in their onset always have had the way prepared for them by processes going on for days, weeks, months, or years. He argues that a depression of the nervous system or clogging of the digestive tract invariably precede disease. By warding off this preceding condition of receptivity, the gates can be closed against the non-specific affections. Rest and dieting are the preventive measures that will ward them off, or attenuate their course if already established. Functional and total rest should be enforced in the early stages of even the simplest affection. Severity of dietetic treatment is the best means at our command to abbreviate the course of any lesion or affection. Instead of wasting strength in digestion, he says, it should be saved for recuperating purposes. Rest and semi-starvation will tide the patient over the "state of morbid imminence." It is especially important for infants, who Londe believes, are habitually fed too much. Even the amounts given in the text-books are too large. Many an infant a year old thrives better on three-fourths of a litre of milk diluted with a litre of water, than on a litre of pure milk. In youth, rest for brain and muscles should always precede attempts at forced feeding. At least, relative rest should always be enforced. In the aged this necessity is likewise imperative. Rest in bed and restriction of the diet once a week or fortnight he considers an excellent measure for the aged and even for all adults.—*Journ. A. M. A.*, vol. xlvii, No. 6.

TRACHEOTOMY.

A Clinical Lecture delivered at the Royal Free Hospital.

By JAMES BERRY, F.R.C.S.

GENTLEMEN,—I propose to speak to you to-day of tracheotomy and to deal especially with certain practical points which I hope may be of value to you in the practice of your profession.

Tracheotomy is an operation which may be required for many different conditions and under widely different circumstances. It may be an extremely easy affair, as, for example, when it is performed on a thin adult with a fairly long neck. On the other hand, when performed, as it so often has to be, on a fat, short-necked infant, it may present considerable difficulties, especially to a young and inexperienced operator. A tracheotomy performed in the operating theatre of a hospital in a good light and where plenty of skilled assistance is available is a very much more simple affair than the same operation performed in a cottage with little or no skilled assistance, in the middle of the night, with such light as may be afforded by a lamp or candle. There are few operations which test the skill and resources of a young operator so much as a tracheotomy performed under unfavourable circumstances. And yet the very nature of the case often requires that the operator shall act promptly and make the best of the conditions present.

Let me advise every one of you to be fully prepared, from the day on which you obtain your medical qualification, to perform tracheotomy on the very shortest notice, whenever you are called upon to do so, to be prepared to do it with very little help from others, to be prepared to do it with very few instruments.

In many cases, fortunately in most, you will have time to make ample preparations for your operation; to obtain the services of one or more skilled assistants; to have ready knife, forceps, retractors, dilators, tube, and any other instruments that you may think necessary. Be thankful when you can do all this. But be prepared to do your tracheotomy, nevertheless, if it must be done, without all these useful adjuncts.

If no surgical instruments at all are at hand, the operation can be done, if it is urgently required,

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with an ordinary penknife, or even a sharp pair of scissors. I have myself seen a tracheotomy done, and done quickly and well, with no other instrument but a knife and a couple of retractors which were made, at a moment's notice, from two hairpins taken from a lady's hair.

Now let us suppose that one of you is suddenly called upon to see a child with urgent dyspnœa the result of some laryngeal obstruction, say acute œdema of the glottis or diphtheria.

The first point you have to settle is whether tracheotomy is necessary or not.

Two points that guide you in your decision are the cyanosis and the recession of the chest-walls. Remember that cyanosis alone is not sufficient indication for the operation. It may be due not so much to laryngeal obstruction as to obstruction within the chest itself. In diphtheria, for example, there may be much blueness of the face from pneumonia even though the larynx be quite free from obstruction. Tracheotomy in such a case as this would be worse than useless.

Recession of the chest-wall is a very valuable indication for the operation. When you see the supra-clavicular fossæ receding deeply at every attempted inspiration, when the lower ribs and the epigastrium are sucked violently inwards each time the child tries to breathe, then the dyspnœa is due to an obstruction in or near the larynx, and relief to the dyspnœa is urgently demanded. I say nothing of intubation of the larynx nor of antitoxin treatment of diphtheria, although both are very good things in their way. I assume for the purposes of this lecture that intubation is unsuitable or impossible in the case before us and that antitoxin treatment is not available or suitable.

The dyspnœa being severe and progressive, dyspnœa and the recession of the chest walls indicating laryngeal obstruction, you decide upon tracheotomy. Now for the operation.

Place your patient in the best light available. You must be able to see exactly what you are doing or you will get into trouble with hæmorrhage. In the daytime place your patient near the window and take care that neither you nor your assistant (if you have one) obstruct the light. At night place your patient if you can on a table immediately under the light if it be a gas-light or electric light, so that the light shines directly downwards into your wound. If you have to utilise lamp or candle-light see that the source of illumination is well above the level of your wound so that the deeper parts of the wound are not left in shadow. A method that may be adopted with advantage when assistants are plentiful is to get some one to hold a large concave reflector on the side of the patient opposite to the light. The forehead mirror of a laryngoscope or the concave metal reflector of a common form Argand lamp serves the purpose admirably. A cone of strong light can then be directed upon the wound itself in much the same way as the limelight from the wings of a theatre plays upon the prima donna.

The next point that demands attention is the fixation of the patient. This is the more important since in many cases it is unwise to place the child fully under the influence of a general anæsthetic. And the difficulties of the operation are naturally increased if the child is struggling.

Movements of the arms and legs may be restrained if necessary by tying them, if assistants are not numerous. But a simpler and better way of completely fixing all four limbs and the trunk without the help of any assistant whatever is the following, which I recommend you to employ whenever you are short of assistance. Place a folded blanket on the table, lay the child on this near the edge of the table. Then fold the blanket towards you over the child and sit on it. The child's limbs are then all firmly wrapped up in the blanket and no movement of them is possible so long as you are sitting on the blanket; and yet if at the end of the operation you want suddenly to do artificial respiration, all that is necessary is to release the blanket by getting up and the limbs are all free again in a moment. With a little care the operator can so arrange his seat on the blanket that he is able to do his operation with ease and comfort.

The head must be steadied by an assistant. If you have but one assistant at a tracheotomy the most important function he can perform is that of steadying the head and holding it exactly in the middle line.

A very important matter is to have a firm support under the patient's neck. In a hospital a sand-bag is a convenient thing to use for this purpose. Any small, hard, cylindrical kind of pillow would do equally well. In a private

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house an ordinary pint bottle wrapped in a towel or inserted into a stocking makes an admirable extempore pillow for this purpose, as I know from practical experience. Unless the neck is supported on some firm resisting structure the neck will tend to fall back into a hollow and the difficulties of your operation will be greatly increased.

Having prepared the skin and having perhaps injected some eucaine and adrenalin (if chloroform is not being given), you are now ready to make the skin-incision.

Take care to make it sufficiently long. It may show great dexterity on your part if you can do a tracheotomy through an inch incision, but it is very unwise to attempt to do this sort of thing. If you are wise you will make a good long incision, have plenty of room to see exactly what you are doing, and then you will not get into trouble with hæmorrhage and everything is likely to go well. But if you have a small deep wound your view will be obscured by blood, you will not be able to see exactly where you are or what you are cutting, and you may end by opening the trachea and pouring enough blood into it to suffocate the patient there and then.

In the case of a fat, short-necked baby the skin-incision should generally extend from the middle or upper part of the thyroid cartilage nearly down to the top of the sternum. In a thin subject the incision may be somewhat shorter, but it is far better to make your incision a little too long than to make it too short.

Too short an incision increases considerably the risks of the operation. The only objection to a long incision is that the resulting scar is a little longer than it otherwise would be. In the case of a life-saving operation like tracheotomy the scar is a matter of very little moment. Besides, it is remarkable how small a scar is left even after very large tracheotomy wounds in children.

Before actually making the incision always place your finger on the cricoid cartilage and make sure that you know where that important structure is.

I have seen a house surgeon (years ago) about to cut down upon the thyro-hyoid space under the impression that it was the crico-thyroid. In a young child the cricoid cartilage is a comparatively soft structure not always easily felt. But the thyro-hyoid space can always be felt with ease, and it is often well to begin by feeling this latter and then to estimate the distance below it at which the cricoid will be found.

In most cases your desire will be to open the trachea at its upper part, if possible between the cricoid cartilage and the isthmus of the thyroid gland. It is seldom desirable, except in special cases, to do a low tracheotomy. The trachea is farther from the surface, there is more risk of wounding unpleasantly large veins, and there is more liability to the occurrence of septic mediastinitis, a fruitful source of death after tracheotomy.

The centre of your skin-incision should be over the spot where you intend to open the trachea, that is, a little below the lower border of the cricoid cartilage. As you deepen your incision through the various layers of the neck, it should become shorter and shorter, so that eventually you have a conical wound the apex of which is at the trachea.

Provided that the head and neck are held exactly in the middle line and that the trachea is not displaced to one or other side (as by an abscess or other swelling), then your operation should not present any difficulty. Keep to the middle line, see exactly what you are doing, keep your wound as dry as possible by clamping or tying vessels as you proceed, and you will not meet with any difficulty. Mishaps during tracheotomy generally occur from undue haste. The operator thinks that if he does not do the operation very rapidly the patient will be dead before the trachea is opened. There are, of course, cases in which it is imperative to open the trachea very rapidly at all risks, but in the vast majority of cases there is quite time to do the operation deliberately and carefully. I do not ask you to waste time over your operation, but I would remind you of the old proverb "More haste, less speed." If, in your hurry, some large vein is wounded owing to your not having seen what you were cutting, more time may eventually be spent over your operation than would have been the case had you proceeded slowly and carefully. I well remember a former teacher of mine who had had a large experience of tracheotomy used to say, somewhat paradoxically, "However bad the child's dyspnœa is, it is probable that it has at least another twenty minutes to live," time enough to do a tracheotomy carefully. What he meant was

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that a child who seemed to the inexperienced to be on the point of death from suffocation often went on gasping for a considerable time before death actually ensued.

So long as breathing actually continues it is unwise to plunge into the trachea regardless of hæmorrhage. It is far better to wait a few minutes longer, to secure all sources of hæmorrhage rapidly but effectively, and then to open the trachea.

The isthmus of the thyroid gland is often a source of annoyance to the young tracheotomist. It may lie just over the place where the operator desires to open the trachea. If the isthmus is narrow and situated low down it can easily be drawn downwards with a hook. But if it is large and covers the first and second rings of the trachea, and especially if that upward prolongation known as the pyramid is present, then it is best to abandon any attempt to draw it down.

Either do your tracheotomy below it or (better, in most cases) lay hold of the isthmus with clamp forceps on either side and divide it boldly. The vessels of the isthmus run mainly transversely and at the upper and lower borders of the isthmus. They can easily be secured. It may be desirable in some cases to dissect out and remove a large pyramid, a proceeding which need not present any difficulty.

Keep steadily to the middle line and gradually deepen your incision until the trachea is reached.

But beware of the retractor of your assistant. It is very easy for an energetic assistant who is retracting the edges of the wound to catch up the trachea and to draw it to one or other side. I have on several occasions seen this cause great embarrassment to the operator. The small and soft trachea of an infant is easily pulled to one side. I have more than once under such circumstances known a house-surgeon carry his dissection down by the side of the trachea. I once had to do a post-mortem on a child on whom the operator had actually succeeded in wounding tne vertebral column before he found the trachea.

Having found the trachea, having laid it bare, and having arrested all but the most trifling hæmorrhage, your next proceeding is to open it. The trachea has been moving up and down with the violent inspiratory efforts and it is well to steady it with a sharp hook if you have one. But often it is enough to draw down the soft parts at the lower angle of the wound and then to plunge the knife into the trachea and cut upwards. It seems unnecessary to state that the trachea of a small child is not as large as that of an adult, but I believe it to be a point which operators in the excitement of a first tracheotomy occasionally forget. An operator whose sole practical experience of the operation has been derived from operating on the adult dead body is apt, at his first tracheotomy, to forget that the trachea of a young infant may be but very little larger in diameter than an ordinary cedar lead pencil. More than once have I known a young tracheotomist to plunge his knife so deeply into the trachea that its point actually wounded the posterior wall. I have seen in the post-mortem room a penetrating wound of the œsophagus produced in this manner.

Be prepared for a rush of air and a splutter of blood and mucus as you put your knife into the trachea, and do not be disconcerted by it. It is well not to have your face immediately over the wound while you are opening the trachea. The patient may cough diphtheritic membrane into your face and eyes, a disagreeable experience which I remember happened to myself at my first tracheotomy for diphtheria.

The trachea having been opened, insert the dilators, but do not be in a hurry to insert the tube. Let the patient cough freely and expel as much mucus and membrane (if the case be one of diphtheria) as possible. Membrane can be expelled much more freely through the open tracheal wound than through the comparatively small lumen of a tracheotomy tube.

I need scarcely remind you that the tube must be put into the trachea and not into the cellular tissue in front of it or by the side of it. The former is an accident by no means unknown. It can easily happen if care be not taken. If air does not pass freely in and out of the tube after its insertion you may reasonably suspect that you have put it in the wrong place. Withdraw it, expose carefully the margins of the tracheal wound, and see that the tube is inserted in the right place.

Tie the outer tube in its place with narrow tape, passing round the neck. Tie it just tight enough to prevent the tube from slipping out. Avoid the elastic bands sometimes supplied for the purpose

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with tracheotomy tubes. Use only inelastic tape, so that the child cannot by hooking his finger under it cause the tube to come out of the wound. I have known such an accident cause the most serious trouble, the tube being replaced only just in time to avert suffocation.

With regard to the treatment of the external wound I would give two pieces of advice. The first is, do not put any sutures into it. Little or nothing is gained by sewing up the wound. Serious harm may result. The wound is from its very nature a more or less septic wound. It is a septic wound involving the cellular tissue of the neck. The safety of such a wound lies in its being an open wound. Again, if the wound be closed even partially at the surface there is a tendency for expired air to be hindered on its way through the wound. It becomes driven into the cellular tissue of the neck and thence is sucked downwards into the thorax at each act of inspiration. Intra-thoracic surgical emphysema occurs and only too frequently proves fatal.

I am fully aware that it is a common practice to put sutures into tracheotomy wounds, and no doubt it is very often done with impunity ; but it is a dangerous practice for all that. I remember once seeing a house surgeon, over whom I had no authority, do a tracheotomy through a small incision. After inserting the tube he sewed up the wound, leaving only just room for the tube. He had made a very neat job of the case and he was very pleased with himself in spite of the scathing denunciation to which I treated him (for he was a friend of mine). But a day or two later surgical emphysema was well established within the thorax and he had to see the poor child slowly die of suffocation which he was powerless to avert ; it had been produced by his own folly.

The other piece of advice which I venture to give you is that it is best to treat a tracheotomy wound with some oily application such as carbolic oil, 1 in 40, rather than with dry antiseptic gauze or with gauze moistened with lotion. It is much more comfortable for the child and is less likely to allow of the occurrence of sepsis, because it prevents the septic mucus and other infective discharges from coming into such close contact with the absorbing raw surface of the wound.

With regard to the removal of the tube let me add that, important as it is in a case of tracheotomy to get the tube quickly into the trachea, it is little less important to see that it is subsequently taken out as soon as possible. From time to time one sees unfortunate children condemned to wear tracheotomy tubes permanently. In many of these cases the child cannot do without the tube, and yet the condition for which the tracheotomy was done was not a permanent obstruction in the larynx (such as papilloma). It was probably an ordinary case of diphtheria or œdema of the glottis. On inquiry you will probably find that no attempt was made to remove the tube for one, two, three, or more weeks after the operation. When the attempt was made, it was found that the child could not breathe without it. So it was left in permanently. Everyone who has had to do with such cases knows how difficult it is to get rid of a tube that has been for months or years in a tracheotomy wound. The practical lesson to be drawn from these unfortunate cases is that as soon as you have done your tracheotomy you ought to begin to be thinking about the time when you should remove it. In some cases, when the tracheotomy is done for a mere temporary obstruction, such as a foreign body or a very acute œdema of the larynx (as from a scald from inhaling steam), the tube may be removed within a few hours.

There are comparatively few cases in which the tube should not be removed, at any rate for a time, by the second day after the operation. There are but few in which it is necessary to retain it longer than four days.

In conclusion, I may sum up the advice I have ventured to give to those about to do a tracheotomy for the first time. I would say to them, keep cool ; do your operation deliberately and without undue hurry ; keep to the middle line and see that the trachea is not displaced from it ; keep your wound dry by tying or clamping vessels as you proceed ; and finally, having got your tube into the trachea, remove it as soon as it has done its work and the original cause for which it was inserted has passed away.

October 1st, 1906.

ACCORDING to MacKee, the beneficial action of the high-frequency spark is twofold, stimulating and bactericidal. Its prime action in the treatment of alopecia rests in its power to produce a vasomotor dilatation.—*Journ. A. M. A.*, vol. xlvii, No. 6.

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A CLINICAL LECTURE

ON

ANÆMIA IN CHILDREN.

Delivered at the Hospital for Sick Children, Great Ormond Street, W.C.

By HUGH THURSFIELD, M.D., M.R.C.P.,
Assistant Physician to the Hospital.

In order to be able to discuss the subject of anæmia in children it is necessary to attempt a classification of the cases which come under our observation. In the present state of our knowledge of the diseases affecting the blood and the blood-forming organs none of the many classifications which have been proposed is wholly satisfactory. We do not as yet know sufficiently the causes or the processes which produce the varying conditions of anæmia so as to enable us to construct a formula which will satisfy both the clinician and the pathologist. The ordinary division, however, into primary and secondary or symptomatic anæmia will serve my present purpose sufficiently well if it be remembered that the division is not a strict one, and that in practice it is not seldom impossible to assign the particular case under consideration to one or other class, and further that it is impossible to assert of the primary cases of anæmia that they will not be in a short time transferred to the secondary class as we get to know more about them.

How, then, are we going to recognise the two different varieties? What is a primary and what is a secondary anæmia? Broadly speaking, secondary anæmia is one to which we are able to assign with a fair degree of certainty an antecedent cause or causes the treatment of which will result in the disappearance of the anæmia. In other words, the secondary anæmias are those about the causes of which we know something, and the primary ones are those about the causes of which we know nothing. But this is not a sufficient distinction: it is too exclusive and too inclusive. There are many cases which are classed among the secondary anæmias although the cause remains obscure, and there are others where we know the cause and yet fail to produce any amelioration of the condition. We must therefore seek another criterion, and that I think we shall find in the results of the detailed examination of the blood. The results of such an examination have done so much of late years towards the elucidation of the problems presented by anæmia that there is a temptation to build up the whole of our classification upon it alone. But very little clinical experience suffices to show that such a method leads to greater confusion even than basing our classification upon clinical methods alone, and the only fair way of treating the subject is to take both the clinical features and the pathological blood-picture into consideration together. Before, then, we can state definitely that a given case is an example of secondary anæmia we must consider the clinical history and antecedent conditions, and have before us a detailed statement of the percentage of hæmoglobin and the number of red and white corpuscles, and in some instances an enumeration of the number of various kinds of white corpuscles which are present.

In order that we may appreciate the meaning of this statement, I must make a few references to the subject of primary anæmias. Of these, two diseases are generally admitted among the profound anæmias, namely, pernicious anæmia and chlorosis. With regard to the second of these, the position is somewhat difficult. My own opinion is that in children there is no such disease—in other words, that chlorosis is a name given to a disease which is particularly apt to attack young girls at the age of puberty and is characterised by a well-known train of symptoms which I need not recapitulate to you, and by a condition of the peripheral blood which is constant in its main features. Those features are a marked reduction in the hæmoglobin, a moderate diminution in the number of red corpuscles and the absence of a leucocytosis of any extent. There is no question that this condition of the blood is often met with in children of all ages, and hence it has become usual to speak of such children as suffering from chlorosis, or from an anæmia of a chlorotic type. But if you look at their clinical symptoms you will find that they differ considerably from those of true chlorosis. The alternative phrase, anæmia of a chlorotic type, may be accepted, but chlorosis simply is, in my judgment, a misnomer, because many of the other features of the disease as seen in young women are absent in children.

For the diagnosis of pernicious anæmia we can

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lay down a much stricter set of conditions. First, the anæmia must be severe, and must be progressive. I am, of course, aware that in pernicious anæmia there are periods of retrogression and improvement, but on the whole the disease progresses constantly to a fatal issue. I should be very doubtful about classing a non-progressive anæmia as true pernicious anæmia. Secondly, the anæmia must present a characteristic blood-picture. The hæmoglobin and red corpuscles must be markedly reduced, but retain their normal ratio, or the hæmoglobin must be reduced to a less extent than the number of red corpuscles; that is to say, the colour index must be normal, or above normal, not below normal as in most other varieties of anæmia; further, there must be no leucocytosis, there must be a marked variety in the shape, the size and the staining reactions of red corpuscles—that is to say, poikilocytosis and polychromatophilia: and, lastly, there must be present nucleated red cells, especially those known as megaloblasts—that is to say, a red cell which is larger than normal, and with a large round or polymorphous nucleus. It is only when all these conditions in the blood-picture are fulfilled that the diagnosis of pernicious anæmia is justified. I insist upon *all*, for each one or several of them coincidently may be present in cases which are undoubtedly examples of secondary anæmia. Thus, one may find marked poikilocytosis in the severe anæmia following hæmorrhage; a colour index at or about the normal in anæmia due to the presence of certain intestinal parasites; and the anæmia due to malignant disease may show a very large number of nucleated red cells and be markedly progressive and yet fail to correspond with the type described in other points. Anæmias which fulfil these conditions are extremely rare in children; in fact, it is doubtful whether more than half a dozen cases which conform to the type are on record. So that although it is necessary to remember the possibility of their occurrence, the presumption in any single case is strongly against the diagnosis of pernicious anæmia.

From what I have said it follows that, with the rare exceptions which have been recognised as instances of pernicious anæmia, children are not affected by the disease. I believe I have seen one case of pernicious anæmia in a child. I may refer to it here. It was that of a child admitted to this hospital seven or eight years ago in a dying condition. His blood was examined, the percentage of hæmoglobin was about 10, there were a million red and about a normal number of white cells—that is to say, 8000 to 10,000 per c.mm. The white cells were present in the normal proportion, and the child died with all the symptoms of profound anæmia. There was no cause found for the anæmia at the post mortem. There was present the Prussian blue reaction which is found in adult pernicious anæmia. The history was that the boy had been ill for two months. That is the only case of the kind I have ever seen, and even in regard to that I felt some doubt whether it ought to be classified as pernicious anæmia. It follows, therefore, that with the exception of pernicious anæmia and with the possible exception of an affection known as the anæmia pseudoleukæmica of von Jaksch, to which I shall refer again, all anæmias occurring in infants and children belong to the group of secondary or symptomatic anæmias.

Having cleared the way, therefore, for a recognition of secondary anæmias, we are confronted by an enormous heterogeneous mass of diseases which are characterised by anæmia. And in order to reduce it to some degree of order we must attempt to make some sort of classification.

The first group, which is readily distinguished, is that resulting directly from loss of blood. In this group it is at once obvious there are two distinct divisions, one where the loss of blood is due to traumatism, the other where the hæmorrhage is the direct result of one or other of the hæmorrhagic diseases. In the first of these divisions the most notable feature is the extraordinary rapidity with which the loss in the corpuscular elements in the blood is repaired. Thus in a case where owing to traumatism the red blood count has sunk to about 50 per cent. of the normal, a period of three weeks to a month is usually sufficient to restore the normal number. At the same time, the new corpuscles are frequently ill-formed, and a large number of microcytes—that is to say, red corpuscles smaller than the normal—are present, and often also a considerable number of nucleated red cells. The nucleated red cell appears with great ease in children in all anæmias, and it does not matter very much, apparently, what the cause of the anæmia is. If the anæmia is sufficiently profound,

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the red marrow of the bones pours out nucleated red cells into the blood, so as to supply the peripheral blood with its corpuscular elements, and the red cell is sent out before it is mature—that is to say, before it has lost its nucleus. The large forms, the megalocytes—that is, cells larger than the ordinary red blood cell—or megaloblasts—that is, the large nucleated red cell—are much less frequent than in pernicious anæmia. There are two other features to which I may call attention, first, that in this as in all forms of recoverable anæmia hæmogoblin increases very much more slowly than the solid elements; secondly, that a leucocytosis of a polymorphonuclear type is a very common accompaniment of the early stages. It is moderate in amount and quickly disappears. So that in any case of hæmorrhage from a severe cut, or hæmorrhage from the bowel for any reason, such as the presence of a papilloma, there is in the first few hours a very considerable fall in the red corpuscles and a still more considerable fall in the hæmogoblin, while after a couple of days the number of the red cells begins to climb up again, but the hæmogoblin remains at a low figure for some time.

In the second division of this first group the characters of the peripheral blood are much more varied. It is obvious that in most of the diseases which lead to hæmorrhage there are present factors which tend to produce an anæmia independently of that caused by the direct loss of blood—or, in other words, the anæmia of this second group is complex. In purpura the anæmia produced by the hæmorrhage is superadded to the anæmia which is a part of the disease. So also in scurvy, and still more in the various types of leucocythemia. However, if we take a case of purpura in which the hæmorrhage has only been trifling, in which petechiæ upon the legs and a small amount of epistaxis are the only hæmorrhagic symptoms, the main loss is in the hæmoglobin, and the corpuscular elements are at or near their normal number. In other words, the anæmia is due to diminution in the hæmoglobin formation, or to destruction of hæmoglobin in the cells without a destruction of the cells. The same is true of infantile scurvy in the earlier stages. In these diseases it is only in the advanced stages or after profuse hæmorrhages from the mucous surfaces that the corpuscles show marked diminution in number. There may be most profound anæmia in scurvy and yet you may find a normal red blood-count; it is the hæmoglobin which is deficient. The question arises whether one can draw useful information from the examination of the blood in such cases. I think the answer is in the affirmative. If in infantile scurvy we find that though the pallor is extreme the corpuscles are present to 70 or 80 per cent. of the normal number, the prognosis is better than it is when the percentage of corpuscles present is 30 to 40 per cent., while if there is a great destruction of red cells as well as of the hæmoglobin the prognosis is much worse. In the case of purpura the deduction must be made with greater caution, because we have less therapeutic control over the disease. Here, again, I should look more favourably on a case where the red count was fairly high than where it had sunk, with the reservation that if the count has been made immediately after the bleeding, such as epistaxis, the conclusion must be delayed until the recuperative power can be estimated. In another disease, hæmophilia, I have no personal experience of the appearance of the blood, but it is usually stated that the characters are those seen after traumatic hæmorrhage. That is to say, there has been a sudden fall in the number of red corpuscles and in the quantity of hæmoglobin, with a rapid recovery in the case of the first and a retarded formation in the second. With regard to the characters of the leucocytes met with in this group of the secondary anæmias, there is little to be said. As a rule, any leucocytosis which is present is moderate in degree and affects the polymorphonuclear leucocytes alone.

The second important group of symptomatic anæmias comprises all those to which we may fairly ascribe a toxic origin. The type is best studied in such a disease as diphtheria, but it includes all the acute infectious diseases which have anæmia as one of their chief symptoms, and such chronic diseases as syphilis and malaria. As to these there is no doubt but when one considers such an affection as rickets one is at a loss whether to ascribe the anæmia to a definite toxæmia or to class it amongst the anæmias of simple malnutrition. The same difficulty is felt in regard to some other diseases, and I do not wish to lay down hard and fast distinctions which I shall be incapable of maintaining in argument.

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In fact, it is probable that in nearly all the secondary anæmias a toxin is at least partially responsible for the blood state, but I shall include in this group of toxic anæmias only those diseases in which the anæmia either appears so rapidly or is so prominent a symptom as to make it all but certain there is a toxin present. Such a disease is diphtheria. In a moderately severe case of this disease pallor is almost always extreme so early as the third or fourth day of the disease. Examination of the blood shows that the corpuscles and hæmoglobin are both diminished to a considerable extent, but that the destruction of the red blood corpuscles is much more marked than in the majority of anæmias belonging to our first group. If an examination is made of the blood of a case of purpura and of a case of diphtheria of equal degree of pallor to the eye it will be found that in the one the pallor is due almost entirely to the destruction of the hæmoglobin, whereas in the other it is due to the destruction of the hæmoglobin and of the red cells themselves. In fact, there is in diphtheria a toxin which actively destroys the red blood corpuscles and the hæmoglobin, producing a very short time an anæmia which is of extreme degree. A similar phenomenon is often observed in acute rheumatism and chorea, especially when associated with endocarditis. The various types of pneumococcus infection act with equal rapidity, and septicæmia and acute nephritis produce the same results. But in all these cases the destruction of corpuscles and colouring matter is accompanied by active leucocytosis of the polymorphonuclear type, which reaches its height in the suppurative affections and in pneumonia.

From these remarks it will be seen that the broad distinguishing feature in this second group is the destruction of red corpuscles and their comparatively slow regeneration, as opposed to their comparative immunity in the first group and their rapid recovery. But though it is legitimate to lay down this distinction dogmatically, I must point out that in both groups there are instances in which the distinction cannot be made. For example, there are many cases of mild diphtheria in which the anæmia is slight and almost wholly due to the diminution of hæmoglobin, the red corpuscles showing little or no change. And in the severe cases of the first group there are instances in which the red corpuscles lose a large percentage of their number and increase again very slowly. In this toxic group there is another feature on which I would like to lay stress, and that is the extreme duration of the anæmia. In many cases, of course, the anæmia rapidly disappears with convalescence, but in some instances it persists for weeks, or even months, without any appreciable alteration. I have at the present time among my out-patients a little girl who had a severe attack of diphtheria more than a year ago who has not yet recovered her normal colour. Her blood is markedly deficient in hæmoglobin, and also, to a less extent, in the number of red corpuscles. In fact, the diphtheria seems to have produced a profound alteration in the efficiency of her blood-forming organs. The same phenomenon is occasionally seen in connection with other infective diseases, though not usually lasting so long as in this instance. In the more chronic conditions which I have assigned to this group the poisons of syphilis and malaria produce an analogous condition of the blood. In both the red corpuscles suffer severely, and in the extreme conditions there may be an appearance in the films of blood resembling closely those met with in pernicious anæmia. In the case of malaria the actual parasite is known and the effect on the red blood corpuscles on which it preys. I have seen only one example in a child, and in this case the number of red corpuscles was reduced to one million and the percentage of hæmoglobin was thirty. In congenital syphilis where anæmia is marked there is a great destruction of red cells, but I have found very slight diminution in the hæmoglobin, a curious phenomenon which I cannot think to be usual.

The third group includes those cases in which there is a prolonged drain upon the strength and nutrition of the patient, such diseases as chronic suppuration, chronic Bright's disease, cirrhosis of the liver, and similar affections. The pallor in these conditions is often extreme, and due to the diminution of red cells and of hæmoglobin. There is, in fact, so far as the blood is concerned, nothing to distinguish these from the preceding or the succeeding group.

These last I have called the group of anæmias due to malnutrition. And as a matter of fact the third group might equally well be said to be due to malnutrition, but in them the malnutrition is due

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to the presence of a particular disease, whereas in the other case the malnutrition is due to the actual inability to assimilate the proper amount of food. It includes the anæmias of chronic wasting, both in infants and in older children—that is to say, the anæmias of indigestion and rickets (which, again, is a question of indigestion) and the anæmias of acute diarrhœal infections. In these the hæmoglobin suffers first and most severely, so that in rickets especially it is usual to find the proportion of red corpuscles about the normal, while the hæmoglobin is reduced to half or one third or even less of its standard.

These four groups comprise a large proportion of the secondary anæmias, but I do not claim that they are well defined. It is often impossible to assign a particular case to a particular group, and there are a considerable number of anæmias which cannot reasonably be assigned to any of them. For instance, a small girl, æt. 4 years, at present in St. Bartholomew's Hospital, is profoundly anæmic, but without any cause of which we have spoken. She had no infective disease, no rickets, no evidence of syphilis or other constitutional defect and no evidence of intestinal parasites, nor was there any history of traumatism; yet her hæmoglobin was reduced to 14 per cent., her red corpuscles to just over a million, she had a slight leucocytosis, chiefly affecting the lymphocytes, a point to which I shall recur; there was scarcely any poikilocytosis, and only very few nucleated red cells. It is obvious that such a case does not correspond to any form of primary anæmia with which we are familiar, and the only group of secondary anæmias to which it could be referred is the toxic group, and there is no evidence of a toxin operating in the case. The child is improving quickly and there is a rapid increase in the number of red cells and a much slower increase in her hæmoglobin. That is to say, on the whole she corresponds to a secondary anæmia of the toxic type rather than to any other. I have a record of two similar cases, but in neither of them was the hæmoglobin so much reduced. But beside cases of this character there is a special type of anæmia met with in infants, and, so far as I am aware, never found in children over three years of age, about which considerable controversy has taken place. This type was originally described by von Jaksch, and was christened by him "anæmia pseudoleukæmica infantum." Apart from its length and the objectionable "pseudo," there is much to be said for the retention of this name. It is at least definitive, which can hardly be said to be true of the name which is more in favour with English writers, "splenic anæmia," for that title, when applied to anæmia in adults, connotes a very different condition. But whatever the name it may bear, the disease itself is interesting, because not only is it of importance clinically, but it has become a sort of centre around which the battles of various writers on anæmia rage. The chief point in dispute is, as to whether it is to be regarded as a distinct disease, a primary anæmia, or whether it is a secondary anæmia, a condition which may ensue in any infantile anæmia. For my own part, I have no hesitation in regarding it as a disease *sui generis*, for the following reasons: First, its strict age limit. It is not met with after the age of three years, and the majority of instances of it are found in children under two years of age. Of course this argument is two-edged, and it may be urged that it is the age which bestows upon the anæmia its peculiar characters. But in answer to this I would point to my second reason, that no case of infantile anæmia has been known to develop into this peculiar condition. All the recorded cases belong apparently to the type *ab initio*. Thirdly, it has clinical characters widely different from those of any other infantile anæmia. A severe rickety anæmia, for example, improves almost at once with appropriate treatment, whereas treatment has little or no effect upon the condition which we are considering. Fourthly, the condition of the blood differs essentially from that of any other form of infantile anæmia. The hæmoglobin and the red corpuscles are both severely affected. There is poikilocytosis and nucleated red cells are present, very often in considerable numbers. There is a considerable leucocytosis of a peculiar type, the chief feature of which is the constant presence of myelocytes in moderate numbers. Clinically the disease attacks infants, producing an extraordinary degree of pallor, a pallor of a peculiar "earthy" type as the phrase goes, so much so that when one of these children comes into the room one can generally recognise it before one is told anything about it. There is a very marked enlargement of the spleen; in fact, the spleen is usually within an inch of the iliac crest, and may reach forward to the

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umbilicus. The course of the disease is chronic, and is practically uninfluenced by treatment. About 60 per cent. of the cases recover, and the spleen slowly diminishes in size, while the blood recovers its normal character. There was a family of children with whom I was acquainted five or six years ago who all had this disease. (Parenthetically it tends to run in families, and, it is alleged, especially affects the Hebrew race.) All the children in this family who had it recovered, and I saw two of them when they were aged respectively five and six. One could still just feel the spleen, but in every other respect they were typically healthy children with a normal blood-count. The odd 30 to 40 per cent. of cases die usually of some intercurrent disease, such as broncho-pneumonia or acute diarrhœa. In the course of the illness punctiform hæmorrhages are usually seen on the legs, the trunk, or face, but there is seldom any bleeding from the mucous surfaces, with the exception of epistaxis, which is not very uncommon in the condition. Post mortem the spleen is found to be enlarged, though usually it is smaller than the signs during life would lead one to expect. The liver is large, anæmic, and fatty, and the lymphoid structures—that is to say, the glands and the follicles in the intestine, and the glands elsewhere in the body—are hypertrophied. Microscopical examinations of the tissues and of the bone-marrow and bacteriological examination have led to no definite results, and on the whole pathological investigations shed no light on the problem.

I will not dilate further on this subject, though its relation to other anæmias is worth a more detailed consideration, especially its connection with true myelogenous leukæmia, but will pass to the consideration of anæmias of children associated with increase in the number of lymphocytes—that is to say, with the occurrence of lymphocytosis. It has often been forgotten by observers who are more accustomed to the examination of the blood of adults than of children that the leucocyte count is normally higher towards the end of the fifth year than in adults, and that the existence of this higher standard is due to the excess of lymphocyte cells in the blood. Hence, at the age of two years it is not uncommon to find a leucocyte count of 15,000 instead of 8000 to 10,000, of which 50 per cent. will be lymphocytes, the polymorphonuclear cells being between 5000 and 6000 per c.mm. as they are in the adult. So in making a blood-count in young children, an allowance must be made for this peculiarity, and if the percentages are always reduced to actual figures it will be sometimes found that the supposed abnormal lymphocytosis practically disappears. Still, there is at times undoubtedly an increase in the lymphocytes in the blood, and in my experience I have found such increase is associated with a more or less pronounced condition of lymphatism, in which the lymphatic structures of the body are hypertrophied—glands, spleen, thymus, lymphoid follicles, Peyer's patches of intestine, and the lymphoid tissues of the naso-pharynx. Such lymphatism in minor degree is very common—in fact, it is seen in a number of ill-nourished children—but in the severe degree it is rare. Another condition in which lymphocytosis is found with some consistency is rickets, though many of the complications of rickets, for instance broncho-pneumonia, tend to produce a polymorphonuclear rather than a lymphocyte leucocytosis, and I do not think that uncomplicated rickets usually presents any leucocytosis at all.

The last group I shall touch on is that peculiar group of which the various manifestations have been called acute lymphatic leukæmia, lympho-sarcoma, and chloroma. Whatever be their real relations—and that they are intimately connected is beyond doubt—they have one feature in common, a leucocytosis, in which the lymphocytes preponderate enormously, being as many as 70 to 99 per cent. of the total number of leucocytes present, that number being from five to twenty times the normal. The cause of this similarity in the blood-picture is obviously the affection of the lymphatic structures, the lymphatic glands, the thymus, and the lymphoid tissue generally. With regard to the factors which govern the varying course of these diseases we are in the dark; but the fact that they are more common in the earlier years than in adults suggests that the adenoid tissue, just as it is apparently at the height of its normal activity in those years, so is at this time most liable to disease, both infective, that is to say acute lymphatic leukæmia, and malignant, that is to say of the lympho-sarcomatous type.

I am afraid these remarks have not done more than classify in an extremely rough way the various forms of anæmia in children, but they will be useful if they serve to impress the fact that there is only one disease, anæmia pseudo-leukæmica, which is peculiar to childhood, that of the other varieties all are met with, not only in children, but in adults, and that the differences between their manifestations in the two classes are due to the normal differences presented by the blood of children and the blood of adults; that is to say as you get normally a different constitution of the blood in the child, so with the same influence at work you will get a different blood-picture in the child as compared with the adult.

October 1st, 1906.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

CASES OF DISEASE OF THE SPINAL CORD.

Delivered at St. Bartholomew's Hospital.

By NORMAN MOORE, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—I am going to lecture to-day on five cases of disease of the spinal cord which are in my wards. They are all cases a little out of the common, and I thought it would be worth while to draw your attention at this lecture to the chief points of interest which they present.

The first is a case of which I cannot say I have ever before seen an example. It is a very uncommon disease. It is a case of the disease known by several names, but perhaps the best known of them is Morvan's disease. Morvan, a physician in Brittany, in the year 1883 first pointed out the characteristics of this particular disease. You have all read in the text-books of the condition known as syringomyelia. You know that syringomyelia is an enlargement, perhaps not always of the original canal of the cord, but at any rate a vacant space occurring in the substance of the cord—that is to say, a prolonged vacant space. There are vacant spaces in the cord produced merely by hæmorrhage, just like the spaces produced by hæmorrhage in the brain which you may find now and then at post-mortems. A smallish hæmorrhage as a rule takes place, the clot in the cavity dries up, and there is a cavity left in which you can find, very often, blood-crystals. A cavity of that nature would not be syringomyelia. Syringomyelia implies a cavity formed in a different way, formed partly, at any rate, in the course of growth, often associated with embryonal tissue. The condition has been known for a considerable time; it was

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known before the time of Morvan, and its prominent symptoms had been described. To put it to you very shortly, its chief symptoms were indications of something out of order in the upper part of the cord; these symptoms were often indefinite, and, therefore, I have put them in this vague way. The symptoms almost invariably pointed to something abnormal in the upper part of the cord, the cervical region, the brachial enlargement, the upper dorsal region. And they were associated often with interference with nutrition, and always with interference with sensation of a particular kind. There was, that is to say, interference with the sensations of heat and cold and sometimes also interference with the sensation of pain. There was very rarely any disturbance of ordinary sensation. Such symptoms were observed in cases in which, post mortem, these vacuities were found in the spinal cord, and the term, syringomyelia "trumpet-shaped hollow," was applied to such a vacuity. Morvan in the year 1883, of course, being well informed on the subject of syringomyelia, in his practice in Brittany observed some cases in which certain other symptoms besides those which I have mentioned were found, and these added symptoms were generally such as seemed to point to an affection of long nerves as well as an affection of the cord. Morvan's papers on the subject were published in the 'Gazette Hebdomadaire de Médecine et Chirurgie' of Paris. He published a whole series of cases, and he himself remarks how very curious it was that in the course of two years he should have collected such a number of cases of a disease which he had never observed before and which had never been described before and that they should have been collected in a single district. His cases are admirably told. This was the first case that he observed, a man called Kerlosquet Serny, obviously a Breton from his name, aged 55 years, whom he saw in 1881. He found that this man had had some pain about his shoulder, but not in the joint, for some time, which he attributed to a fall. The pain was moderate, but was followed by an incomplete paralysis throughout the whole of the right arm—that is to say, the arm on the side on which he had the fall. During the five or six years following this gradually grew somewhat worse, and when Morvan saw him he found that both biceps, triceps, and extensors were unable to work with proper energy, though not absolutely paralysed; that there was paralysis of feeling in the whole of the left arm—that is to say, the opposite arm, and there was a slight trace of commencing paralysis of feeling there too. He found that this referred chiefly to temperature and pain sensations. So there was analgesia, but no true anæsthesia. Sensibility to touch was preserved everywhere, and even when the eyes were covered he could point with precision to the point at which he aimed. Morvan found that at uncertain intervals he had had sores on the extremities of different fingers. One was the seat of a whitlow, and he lost during the winter the ungual phalanx in consequence of this. He did not suffer in the least by the incision necessary to remove the phalanx nor by the extraction of the decayed bone, it did not cause him any pain whatever. During a series of weeks Morvan published a number of these cases, but that was the first which he observed.

Our patient shows symptoms of this kind. He is a builder's labourer, aged 29 years, and complains of loss of power in the right hand, and he has imperfect grasp in that hand. He has suffered before from frontal headache; he has had no very marked symptoms except those of the hand. Six years ago he had an injury to the right elbow, with separation of the external condyle of the humerus. In many of these cases there is some history of injury of this kind, and it seems possible that the disease itself may have something to do with an accident in some instances. In September, 1904, he had a whitlow on the first finger of the right hand which was painless, and eventually, after many unsuccessful attempts at healing, the finger was amputated at the first interphalangeal joint, without an anæsthetic and without causing him any pain. Gradually the fourth and fifth finger became flexed, and he lost power in his arm. He then had several more whitlows on the same hand. Each time he has had a whitlow he has had pain across the back in the same spot, about the second and third lumbar vertebræ, a very interesting circumstance in relation to the certain existence of disease in the spinal cord. He complains of a feeling of cold on the right side. My colleague, Mr. Rawling, who very kindly drew my attention to this patient some months ago when I was lecturing on syringomyelia, treated his hand surgically. The patient's knee-jerks are exaggerated, there is no ankle-clonus. His sensations to touch and pain are very much

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interfered with. I will show you a chart which my house-physician, Dr. Hine, has drawn illustrating that. In the same way the sensations to heat and cold are profoundly affected. (See charts Nos. I, II.)

The patient's condition has not altered since admission. On the middle finger of the right hand, in the first phalanx, there was a slight suppurating ulcer and a small blister at the tip; the finger was opened, and healed without any pain. On January 31st the report was: His hand is worse to-day, supra-trochlear gland and some glands in the axillæ enlarged, no pain. The hand was opened by an incision in the palm along the tendon, one and a half inch above the metacarpo-phalangeal joint. Another opening was made above the level of the unciform process, and drainage established by gauze between. All this caused no pain whatever.

Here are the two excellent charts. One shows very well the interference to touch and pain sensations, ordinary sensation remaining over a large part of them unchanged. The other shows the loss of sensation to heat and cold, and the part where he feels heat and cold as cold, the part where he confuses heat and cold, and the part where it is less impaired. The loss of sensation of heat and cold, while common sensation remains, of course is due to the fact that those sensations are probably, we do not know for certain, conveyed through the periendemial tissue by the dorsal roots, past them to the brain, whereas the part further back still conveys common sensations. The patient does not feel anything at all on the right side. In the left arm, which is the one less affected, he feels the hot tube and the cold tube—which is very distinctly cold—as exactly the same. It is characteristic of Morvan's disease that it begins upon one side, that it often extends to both, but that for a long time it is much more marked on one side than on the other. In this case you see it is marked on the right side. The loss of power in his arm shows that the nerves are to some extent affected as well as the cord. So that this case, as are all those of Morvan's disease, is to be regarded as a case which is primarily spinal but in which the peripheral nerves begin to be affected. Such are the chief points about this very interesting and very uncommon case.

Now I come to these two patients. This first is a case of transverse myelitis. I will read the account of it. He was a ship's sick-berth steward, and is aged 35 years. On August 15th, 1905, he found he had to drag his right leg when walking. The next morning he woke up and found he had lost control of his bladder. At the same time he found that his left leg was becoming weak. Forty-eight hours from the first onset, on August 15th, he had complete loss of power and sensation in both legs, complete loss of power in both sphincters. During the first month of his illness he was troubled with constant sickness after food. Up till April in this year he had no feeling in his legs, but in that month electrical treatment was applied, and he experienced tingling in the toes of the left foot and was able to move the left foot and the toes of it. He had syphilis thirteen years before, and had been treated for some time—on and off for four years. On admission he had no control over bladder or rectum. His mental state is normal. He moves both his arms perfectly well. The sight is quite good. Below a line corresponding to the ninth dorsal vertebræ there is partial anæsthesia to touch sensation. His temperature sensation is absent. The legs are rigid, especially the left. You see it drawn up now and rigid, and with such rigidity that it is almost impossible to get the knee-jerk. I cannot extend the legs by the use of any justifiable amount of force. The knee-jerks, when one succeeds in getting them, are exaggerated. He has no ankle-clonus and the left ankle is generally rigid and extended as it is now. He has no bed-sores. At one time he had one or two, but that was before he came here. If his bladder is allowed to fill it overflows, but after using the catheter he can retain his urine three to four hours. There was some cystitis, which I hope is now diminished. He has now some sensation in the rectum and can feel the passing of a catheter. He can force his knees down by means of his leg-muscles, but they are only slightly improved. His cystitis is much better. Therefore this is a case in which sensation has improved. The defective temperature sensation and the general loss of sensation was a transitory condition. The complete loss of muscular control represents what one may call the permanent degree of the lesion. May it be reduced? Of course, we hope it may, but it obviously marks the extent of the lesion. That, then, was a transverse lesion. The upper part of the patient is free from all morbid condition, but the legs, bladder, and rectum are paralysed. There is a sign of irritation, perhaps degeneration, in the rigidity of

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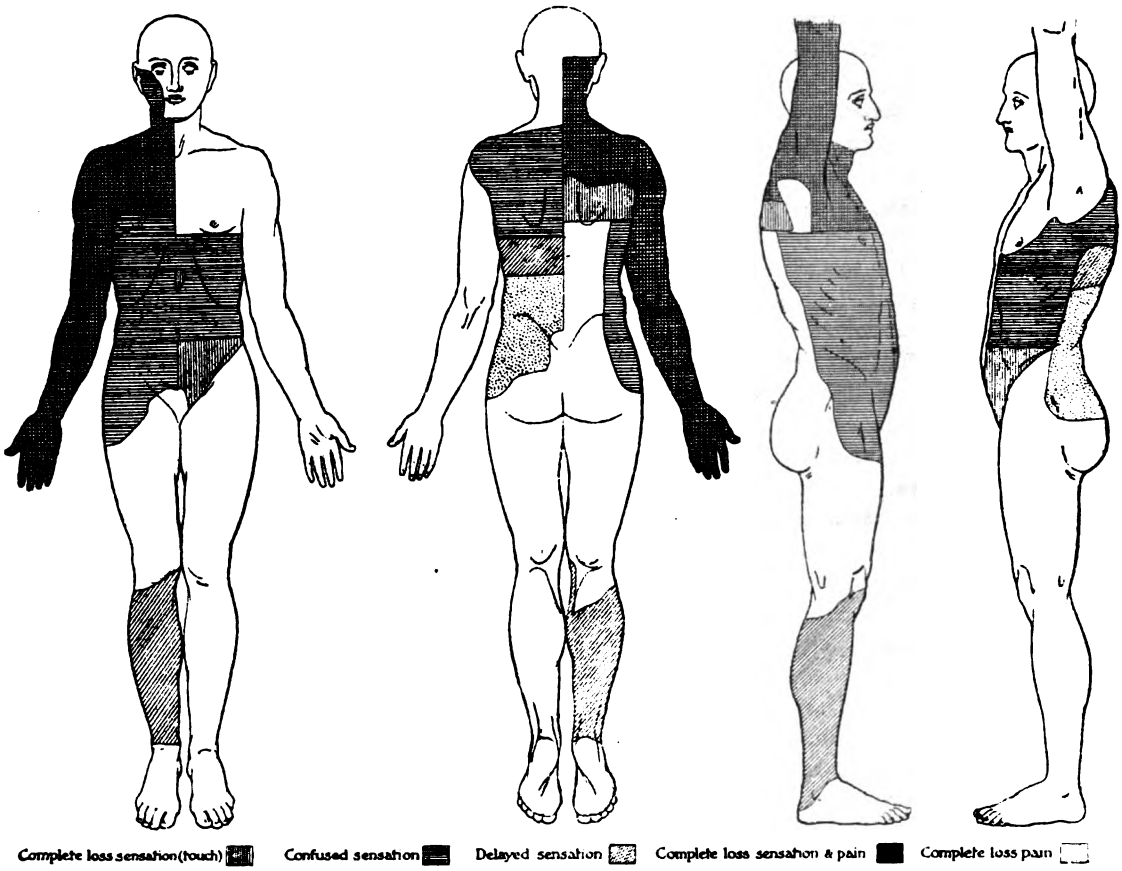
his legs, but it is altogether below a certain level. There is no impairment of nutrition of those muscles, there is no marked impairment of electrical reactions. Notice how very quickly the whole thing came on—in about forty-eight hours or a little more. That was indeed an acute transverse myelitis. How does such a myelitis originate? One could easily believe that it might originate by an injury; but there is no history of injury. One has seen cases—and probably you have all seen them in the surgical wards—in people who have been run over and had a very similar condition to this. Of course, disease of the vertebral column would produce such a condition, but if a man had that condition—that is to say, one of the vertebral column—as long as this patient has had it (since August, 1905) we should have discovered the morbid condition of the spinal column. So I think I am right in believing that none is present. We know very little about the causation of transverse myelitis of this kind. There is no doubt it is commoner in people who have had syphilis, and that it appears to have some relation or other to some anatomical condition produced by syphilis. What is that? Most probably a vascular condition, a thickening of the arterioles, and so interference with nutrition, which goes on till it finally reaches the point which produces the symptoms. Of course, that sort of result with vascular disease we are familiar with in the brain. You often see cases which you examine post mortem with great thickening of arterioles, and with large arteries and arterioles and consequent degeneration of the nerve tissue, which must have been going on for a long time, although the sudden seizure and the consequent paralysis was a matter of a few hours. It is possible something of that kind may have been the cause here. It is a perfect example of transverse myelitis.

CHART I.



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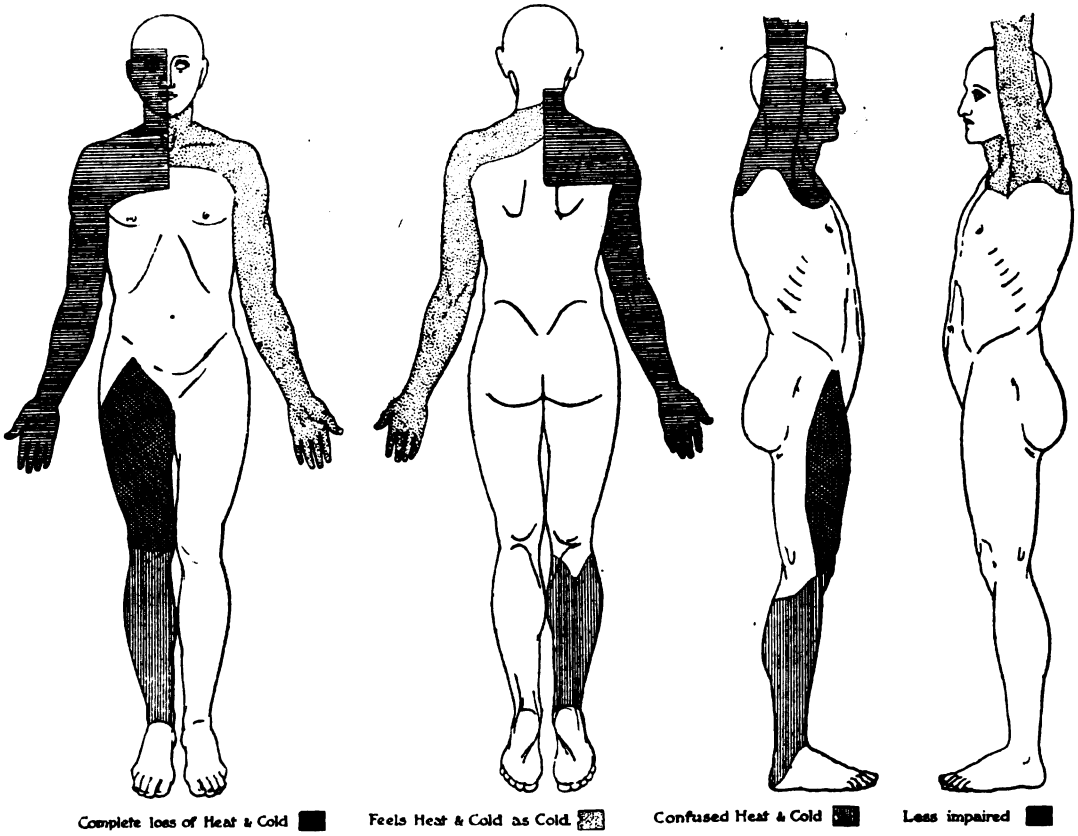
The next case is a very interesting contrast to that. The patient is a man from Hampshire, aged 20. He has loss of power in his legs. He has a little power remaining, but he cannot raise his legs. His arms are more or less affected. He grasps fairly well. He has some loss of power in one arm. He has now complete control over his bladder and rectum, and they have not been affected. There is another difference—namely that his legs are not rigid at all. There are no knee-jerks, and his legs are flabby, and if not kept artificially warm they rapidly get unnaturally cold. So there clearly is an interference with muscular nutrition in several of the muscles of his legs. He is employed on a farm. I will read you a history of how his illness began. It was scarcely less sudden than the last one, and perhaps in some respects it was more sudden. He had always been well and was of temperate habits. On October 13th last, on trying to get up he found he could not put his socks on without pain. The pain was of an aching character across the abdomen. He got up and dressed and walked three miles. He went to bed that afternoon as he felt so ill, and has not got up since. On the next day when he tried to get up he could not do so. The following day he could not use his legs nor move them, and his right arm was as bad. His left arm he could move, but was unable to feed himself with it. He had a zone of pain about three inches broad across the abdomen, through the umbilicus, and became semi-conscious for a week. There is no sign in him whatever of brain disease; he is absolutely intelligent. There has never been any brain disturbance. That semi-consciousness is merely one more illustration of the fact that a very severe nervous lesion has effects in the nervous system far beyond its anatomical area. It was part of the general disturbance of his system. And when he became sensible he made no improvement for a month.

CHART II.



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He never lost sensation. He can feel my hand on his legs.

Now listen to the account of his electrical reactions given by Dr. Lewis Jones. They are most important. Remember that in the other case there is no marked change in this respect. In his arms there are traces to the coil. The extensor communis digitorum is affected least to the cells. There is sluggish contraction of poor quality above the elbow and forearm. The flexors are normal, the extensors of the thumb almost normal, and in the extensors of the arm the reaction is almost absent. In the left arm to the coil the reaction is normal below the elbow, and above it is sluggish to cells and coil. In the legs both interossei are normal to the coil. The right anterior and peronei react very faintly to cells plus current. Calf, nothing. A faint contraction in the quadriceps and adductor magnus. Left, sluggish to cells; nothing above the knee. Gluteus reacts a little to + cell. His hamstrings and adductors give well-marked reaction of degeneration. The quadriceps give a faint flicker of the reaction of degeneration on the left, nothing on the right. The leg-muscles all show the reaction of degeneration markedly. The interossei show the normal reactions—especially good on the right, weaker on the left. There is reaction of degeneration of the levator ani, recti, and probably obliqui also; there is nothing abnormal in the glutei. So you see we have here well-marked reaction of degeneration in several muscles, but nothing of the kind in the other patient. There is a tendency in this patient to grow cold, and a feeling of impaired nutrition instead of the rigidity which is obvious in the other man. This, in fact, is a case of poliomyelitis, and a very extensive example of what we commonly speak of as infantile paralysis. You have all seen, in the out-patient room, a child with one leg wasted and cold and hanging quite limp, and you find, on going into the history, that the child had an acute illness, which lasted perhaps a week, and that in the course of this illness it had, at a date which could not be fixed because the child was then in bed, but probably very soon after the beginning of it, paralysis of its leg, which has remained. That condition, on an exceptional scale, is shown in this patient. He clearly has degeneration of cells of the anterior horn, producing a profound atrophic lesion in addition to the paralysis. It is a very uncommon example in two ways—one, in the widespread extent of the lesion and its consequent effects, and secondly, in the age of the patient. Such lesions are much commoner at an earlier age. Those two cases, therefore, afford a most interesting illustration of the difference between the results of an acute transverse lesion and the results of an anterior polar lesion.

The two remaining cases are both examples of obscure general conditions belonging to the locomotor ataxy group of diseases. This patient walks quite well. He can stand fairly well with his eyes shut. I cannot get any jerk in his right leg, but it has been obtained. There is no jerk in his left leg. In the right it is certainly much diminished. The pupils are small. He has pains shooting down his legs. So you see that of the symptoms of locomotor ataxy he has absence of knee-jerks, contracted pupils, shooting pains down his legs. His age is thirty-four and he is a porter. Ten years ago he had syphilis, was sent to the Lock Hospital, was treated for a month and did not go back again. Therefore probably the disease was not eradicated. He first noticed stiffness in his right thigh ten months ago, which became worse despite treatment. The left hand was then affected by paralysis. He has wrist-drop on the left side. He cannot raise the left hand, but he can raise the right. Here in his right hand he has well-marked wasting of the thenar and hypothenar eminences, particularly of the finger, and here, again, on the left there is similar wasting, though not so great as in the right. The pupils react only sluggishly to light and accommodation. He has no facial paralysis and no true ataxy, and no Romberg sign. On the left there is a pronounced wrist-drop, with obvious wasting, and the grip is feeble. In the right side there is pronounced wasting of the finger muscles and the grip is feeble. There is a supinator jerk present, but there is no triceps jerk; the sensations are unimpaired, the electrical reactions of both supinator longi are good. In the left arm there is no contraction of any extensor except the carpi ulnaris and of the thumb, showing how profound the extensor paralysis and the impaired nutrition of the left arm are. That is a case, I believe, of locomotor ataxy, or tabes dorsalis. No doubt it is true that the man has not the disease to such an extent as to show it in his gait, but there is an

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absence of reflexes, he has distinct lightning pains down his legs, and has great impairment of his pupil reactions. Added to that is this condition, and I chiefly wanted to draw your attention to him to remind you of this fact, that locomotor ataxy is often associated with additional lesions of different parts. Here is a case in which the additional lesion, which has lasted a much shorter time than the others, is a muscular atrophy affecting the extensors on the left side and affecting the finger muscles on this side. Now, are the two conditions entirely separate, or is the man really suffering from one continuous nervous disease, due originally to his syphilitic infection? I am inclined to the view that this is the true explanation of his condition.

The case of the other patient is one in regard to which I had very great difficulty indeed in coming to an opinion. The patient is a man aged 57 years, who says that twenty-five years ago he was in Guy's Hospital with locomotor ataxy, and was an out-patient there for two years. For sixty times he was suspended by the head from the ground. That was then a method of treatment on its trial, and he thought he benefited by the treatment. He had suffered much from stabbing pains in the leg, and had an attack of abdominal pain and vomiting. To relieve this he took large quantities of whiskey and occasionally morphia. A month before admission an ulcer had begun to form on the second toe of the left foot, about as large as a sixpenny piece. It was always painless. There was a history of syphilis and of previous taking of morphia. This man had permanent and well-marked contraction of his pupils. With that history, and as he was able to walk, and the signs of ataxy were extremely indefinite, for some time I was inclined to the view that his pupil condition was due to the morphia which he had taken. But we satisfied ourselves that he was not taking any morphia in the ward; it was inaccessible to him. He began to sleep pretty well, and I think it was quite clear he was not suffering from morphia-poisoning. But there was some other condition affecting him. When one came to add up his symptoms one came to this: he had a gastric pain which clearly had nothing to do with food, and was entirely separate from gastritis. There was pain across the upper abdomen, pain in his legs, though it was difficult to be certain whether those were true lightning pains. They extended down his legs and he said they were worse in cold weather. Very likely they were lightning pains. The pupils were invariably contracted. He had no knee-jerks. On the plantar aspect of his second left toe he had an ulcer which was unhealthy-looking, of the perforating type, just like the ulcers on the feet of people who have locomotor ataxy, and which very rarely occur from any other cause, or associated with anything else than the impaired sensation of that disease. But there was this point of difficulty: the proximal phalangeal joint of his toe was clearly not natural, and it seemed just possible it might be associated with this ulcer. However, his ulcer was properly treated and watched and got quite well. So, taking that into consideration with the other symptoms, I believe that it was a true perforating ulcer, and not a small outside manifestation of the disease of the joint. His pupils remained contracted. His knee-jerks never appeared. We did not see him in any attacks of pain, but we accepted his account. That was a case, of which you are sure to see many, in which you long remain in doubt whether the patient has true tabes dorsalis or not. The only thing you can do in such circumstances is to collect the evidence, and where you find a perforating ulcer of that kind, with absent knee-jerks and contracted pupils which do not react to light—his did not—you will be correct in believing that the patient has locomotor ataxy, slight in degree and slow in progress, but which if he lives long enough is likely to develop further indications of the disease.

Let me just recapitulate, then. The first of those four cases was of that variety of syringomyelia called after its first describer Morvan's disease, and the characteristic which distinguishes it from syringomyelia of the ordinary forms is the presence of painless whitlows on the hands. The next two cases illustrate the difference between acute transverse myelitis and acute anterior poliomyelitis. The remaining cases were examples of two varieties of locomotor ataxy.

October 8th, 1906.

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WITH MR. PEARCE GOULD IN THE WARDS OF MIDDLESEX HOSPITAL.

(1) *A Case of "Hysterical Paralysis" of the Left Arm.*

THIS patient, a girl æt. 18 years, came in with a history that six months ago she fell and "dislocated" the left shoulder, and of there having been two "reductions" under an anæsthetic. There was complete paralysis of the left upper limb; when she came in she could not move a finger or contract any muscle in the left upper limb. You will remember that I pointed out on a former occasion that there was no wasting of the limb, in spite of the six months' paralysis. We had the electrical reaction of the muscles tested, and this showed a normal response to faradism. For obvious reasons I do not intend to mention the diagnosis in her presence. We have applied faradism daily during the last week, and told her she would certainly get well, and that probably the restoration of the use of the part would come to her quite rapidly. You will see now that she can lift her left arm to the fullest extent and can put it behind her waist. If I had mentioned "hysteria" in her presence, or *mal imaginaire*, or anything of that kind which she could understand, we should most probably not have had this result. We have carefully avoided any suggestion of "hysteria" to her, and she has not been allowed to have any idea of the diagnosis. The diagnosis was arrived at, as you know, by noticing the entire absence of wasting and of all trophic changes in the limb after six months of total paralysis, and by the normal reaction of the muscles to the faradic current. We were struck also by the complete paralysis of the whole limb after a comparatively slight injury—a paralysis that would have indicated the laceration or very severe contusion of the whole of the three cords of the brachial plexus, or of all the nerves arising from them, if it had been organic. Of course there is more than one way of dealing with cases of hysterical paralysis. One is the method which we followed here, namely to take care that the patient is not allowed to have any notion of the view taken of the nature of her malady, and to apply the faradic current to the paralysed muscles, and to combine with this an assurance of speedy and probable sudden recovery. There are several points in favour of the use of electricity. It is a definite application of something which has an interest for the patient; it is novel to the patient; it is accompanied by a certain amount of noise, and the faradic current causes a pricking sensation; and then there is the response and the twitching of the muscle to which the electricity is applied. All these factors combine to excite the patient's interest in the treatment, and this is distinctly helpful. It is certainly true also that an assurance that there will be recovery, and that it is very likely to occur suddenly, will help that very thing to occur. It was so here. She has made a very rapid recovery. It is proposed to carry out the same kind of treatment in the convalescent home to which she is to be sent, and I think we may rest assured that in a month's time she will be quite well.

(2) *A Case of Calculous Pyelonephrosis. Nephrectomy on the previous afternoon.*

This patient is a woman, æt. 50 years, who was the subject of pyelonephrosis. You will remember the history, as we discussed this case before the operation. Ever since the age of seventeen she had had pain in her right loin, and latterly she had noticed a tumour develop; for a long time she had noticed that her urine was "thick." When she came in she had a large tumour in the right loin, and there were both pus and blood in the urine. We concluded that she had a large stone in the kidney, with extensive pyelonephrosis. In that belief I operated upon her yesterday, and the operation confirmed our diagnosis. Now, I want to ask you how you would have operated. You would have made a posterior incision parallel with the last rib. As it was a right pyelonephrosis you would place the patient on the left side with a pillow beneath the loin so as to bulge up the right loin. A convenient place for the incision would be one inch below the rib. Remember, you want your incision to be high up rather than low down, because the bulk of the kidney is above the last rib. The incision should reach backwards to the outer edge of the erector spinæ, and anteriorly to a length corresponding with the condition for which you are operating, for 4, 5, 6, 7, or even 8 inches. If you are to remove a big solid tumour, or want to follow down the ureter towards the bladder, you

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will make a much longer incision. Here in this case we made an incision 5 inches in length. In making this incision what should we come upon? We cut through the skin and superficial fascia and then the deep fascia, the latissimus dorsi, the posterior portion of the external oblique, and then saw the posterior tendon of the transversalis abdominis. I made a little cut in this parallel with its fibres and enlarged it by tearing, so as to split the aponeurosis rather than sever it. Underneath that was the transversalis fascia passing backwards to the spine. Between the posterior aponeurosis of the transversalis muscle, often called the lumbar aponeurosis, and the transversalis fascia there is a layer of fat that bulges up into the incision when the aponeurosis is split, as it did in this case yesterday. Having torn through this fat, one came upon the transversalis fascia, and tore through that, and then came down upon the actual perirenal or subserous tissue. In some cases you will find the relative thickness of those two layers of fat very different. Yesterday we found the superficial layer underneath the fascia lumborum very thick, and that immediately about the kidney very thin—hardly any fat in the cellular tissue. In some cases you will find this cellular tissue very fatty indeed. It is not difficult for us to see why the fat had gone in this case. Remember, the kidney was greatly distended, and no doubt it had been so for a very long time, probably for years. During that time there was pressure on the kidney, and that pressure would first of all tell upon the fat immediately behind it. It was that fat which was absorbed, so that the fibrous capsule of the kidney was brought almost absolutely into contact with the fascia transversalis. After tearing through this thin fascia, one had exposed the kidney, which appeared as a big cystic tumour bulging into the wound. Obviously my next duty was to empty this cyst. By means of a trocar and cannula we drew off 34 oz. of fluid of a pea-soup consistency. After the cannula was withdrawn a few more ounces escaped, so that altogether nearly two pints of this thick, purulent fluid was taken away. The house-surgeon, who had been supporting the tumour from the front, felt the tumour gradually disappearing under his hand. Towards the termination of the evacuation of the cyst the point of the cannula grated against a stone, and thus one was able to confirm one's diagnosis of stone as the cause of the trouble. The collapsed cyst was carefully separated from the parts around, the renal vessels were securely ligatured, and the tumour delivered and removed. Is there any particular precaution to be taken in delivering such a tumour as this? Yes. In the first place, it must be very gently separated, so as to avoid tearing through the peritoneum. This kidney had probably been the seat of a stone ever since the pain which occurred when the patient was seventeen years of age; that is to say, she may have had a stone in the pelvis of the kidney for thirty-three years, and latterly it had become impacted in the pelvis of the kidney. The result was that there was no kidney-tissue left that we could see with the naked eye. We must remember that a tumour forming in this way immediately under the peritoneum may be adherent to the peritoneum in front, and it is quite possible to tear the peritoneum in separating such a tumour. Of course we are not so afraid of wounding the peritoneum as we used to be, but it is desirable to avoid tearing it, and one would not think of running the risk of contaminating the peritoneum with the material which came away from this kidney. Another precaution is to avoid injury to the colon or the duodenum. In some cases the colon becomes firmly adherent to the kidney, and you have to be very careful not to damage it. The duodenum is in front of the hilum of the kidney and to the inner side, and it is when you get round to the hilum that you think of that. We should separate off the suprarenal capsules inside. Another possibility is that I might have injured the pleura. One may injure the pleura in two ways, for the pleura may extend below the last rib, and you may injure it in the initial incision. We are speaking, however, of peeling off the tissues from the kidney in delivering it, and that is when the pleura is more likely to be injured, because the kidney may be firmly adherent to the diaphragm, and you may tear away a bit of the diaphragm. That is another reason for exercising great gentleness and being in no hurry in separating the kidney tumour from its surrounding tissue. Another point to remember is that the liver may be injured. But a more important possibility than any of these is that you may tear the inferior vena cava. Sometimes one has to remove a kidney which is very firmly adherent indeed, and then the separation of it from the surrounding structures is a matter of considerable

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difficulty and some anxiety, because there are such important structures in its vicinity. In this case we had no difficulties of that kind. We gently peeled the tissues off, keeping closely to the kidney itself. When we came to the vessels we tied them, and there was nothing further necessary. It was a simple, straightforward case. There is a method by which you can render the separation of the kidney safe from injury to the adjacent parts: it is by separating the kidney from within its fibrous capsule. We could not apply it here, for the kidney was so distended that if I had attempted to separate the remains of the fibrous capsule of the kidney from the rest of the cyst wall I should have torn into the cystic cavity and made the rest of the operation much more difficult. We made sure that our ligatures were secure and that the wound was dry, and then I closed the wound. I put a large split drainage-tube with a wick in the posterior corner of the wound, and closed all the wound in front of it. To close the wound it is best to unite the severed muscles and lumbar aponeurosis by buried sutures, and then use silk-worm-gut sutures for the skin.

And what do you want to know about the patient to-day, the day following the operation? You will ask about the temperature. It is 98·2° F. Then the pulse and respiration require to be looked to. The pulse was 88, running up to 100 this morning, and the respiration rate 20. You will next ask whether she has passed any urine. If she has not passed her urine you will ask whether a catheter has been used. Your object is to know whether urine has been excreted into the bladder. We learn that at 4 a.m. the catheter was passed and 6 oz. of urine were drawn off. At 10.30 she passed 4 oz. and at 12.30 4 oz. That is to say, she has excreted 14 oz. since the operation twenty-three hours ago. That is fairly good. The urine has been examined and is found to contain neither blood nor albumen. It is strongly acid and the specific gravity is 1030. It is dark in colour. This quantity of urine is much less than the normal secretion—50 oz. in twenty-four hours.

How, then, shall we account for her having passed only 14 oz.? In the first place she has had much less than the normal quantity of fluid. She was given no fluid after 10 a.m. yesterday morning, and before she was taken to the theatre at 2 p.m. she emptied her bladder, so that we started with a "clean slate" in that respect. And then after coming back to the ward she was for several hours without fluid. Then, again, she was sick last night, and you have to allow for the fluid which she vomited. So that the fact that she has excreted 14 oz. of urine in twenty-three hours, especially when we remember that she has vomited more fluid than she has taken, shows that her other kidney is working well. And in such a case, shortly after the operation, you would, of course, enquire whether the urine contains blood, or pus, or albumen. You will also ask her how she "feels," as you say, but there is something more important than that. Supposing one of the ligatures had been imperfectly tied, or had slipped. That would be very important. And so the first question I should ask the man who dressed this case is, whether he had to dress the wound because of bleeding, or whether when he dressed it he found that there had been undue hæmorrhage. So hæmorrhage would be one complication which would be inquired into. We hear that it was dressed at eleven o'clock this morning, and that it did not require to be dressed last night. There was a moderate amount of blood and serum in the dressing. What else is there to ask about this morning's dressing? Let us look at the matter in another way: what might have gone wrong with the case? There might have been a good deal of hæmorrhage. Or we might have infected the wound at the operation; and by this time the edges of the wound would have been reddened and tense, and this would be accompanied by a rise of temperature and a more frequent pulse. We must also enquire how she bore the shock of the operation, how she recovered from the anæsthetic, and satisfy ourselves whether she has any signs of commencing peritonitis.

These are six questions which we want answered about her at this stage. The temperature-chart answers some of them. She does not seem to have suffered from shock last night, as the temperature was 98° F. and it was 98·4° F. this morning. So that one may say she quickly recovered from the shock. She has been sick once, but all chloroform sickness seems now to have passed off. The other kidney has been doing its work well; there has been no excessive hæmorrhage, and we have now remaining the question as to whether there is peritonitis. We want to know whether there is any abdominal tenderness. There is tenderness

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on putting the hand on the right side, but there is apparently no general tenderness and no rigidity. Immediately I put my hand on the right side of the abdomen she gave a deep sigh and her abdomen moved very freely indeed. There is no distension whatever. That deep abdominal respiration on putting the hand on the abdomen told me plainly that there is no peritonitis, and this view is supported by the temperature, the pulse rate, the absence of sickness, and the respirations at 20. She is not absolutely out of danger, but "well begun" in these cases is more than "half done."

(3) *A Case of Appendicitis in a Woman seven months pregnant.*

The particular interest of this case lies in the fact that the patient, a woman nearly seven months pregnant, has been admitted with symptoms of acute appendicitis. It is not a very common combination, and there are certain important questions which arise in connection with such a case, so I thought it would be well to draw your attention to it. Let us ask ourselves two questions, first, How will the fact of a woman being pregnant affect the course of a case of appendicitis? and secondly, How will the existence of appendicitis affect the course of pregnancy? Picture to yourself an abdomen which, instead of containing intestines and an inflamed appendix only, has a big uterus rising up and pushing the intestines up. Of course there will be pressure on the appendix and the inflamed area, and it is believed that the presence of a big uterine tumour may stretch and tear across adhesions and so lead to the diffusion of a previously circumscribed peritonitis. So that instead of having a localised peritonitis, it may become rapidly diffused, and an abscess become internally ruptured and the pus escape into the sound peritoneal cavity and cause widespread diffuse peritonitis. And the fact that a woman is pregnant, especially in the later months of pregnancy, renders her rather more vulnerable—less able to withstand the ill effects of appendicitis. The prognosis of appendicitis is therefore not so favourable in a pregnant woman as in the non-pregnant woman. But the serious part of the co-existence of pregnancy with peritonitis is the effect which the peritonitis may have on the course of the pregnancy. There have been many cases of appendicitis in pregnant women in whom the pregnancy has run the normal course. Where that appendicitis has been plastic and has subsided, or where an abscess has been opened and drained, the pregnancy may go on to its natural termination. On the other hand, there is great danger of miscarriage or premature labour. The appendicitis may terminate the pregnancy simply by "irritation," or by the inflammation spreading to the walls of the uterus and exciting irregular contractions of that organ, and altering the metabolism of the uterine wall altogether. But appendicitis may affect the course of pregnancy in other ways than this. The patient is ill; toxines are absorbed into her blood, making the patient ill, and these toxines may kill the child. We have seen cases in which those poisons have killed an adult, and therefore it stands to reason that the feeble life of the fœtus may much more easily be terminated by the same poisons. But the disease is often virulently infective, and as a result of this the Fallopian tubes may become infected, then the uterus and especially the placental site may be infected, and the infective organisms have even been found in the viscera and blood of the fœtus. To sum it all up, then, while appendicitis may have no injurious effect upon gestation and the pregnancy may go on to full term, yet, on the other hand, it may kill the fœtus and thus terminate pregnancy, or, what is worst of all, it may infect the uterine walls, the placental site, and lead to acute metritis, perimetritis, parametritis, and general blood-poisoning—in one word "puerperal fever." It comes to this, then: while pregnancy makes appendicitis worse, appendicitis exposes a pregnant woman to special dangers and very serious complications. So you have to look at these cases with very great care indeed. If it is a generalised peritonitis there is never any time to wait, whether there is pregnancy complicating the case or not. And, if I may use such an expression, there is even greater urgency in operating if the peritonitis is accompanied by pregnancy. Where there is abscess you will do as little as you can; make a free incision into the abscess by the shortest route, and drain it. Do not make a prolonged search for the appendix, but get rid of the infective pus. Sometimes the question comes before us in quite a different form. A young woman who is a couple or three months pregnant has a slight appendicitis, perhaps one of a series of attacks, and the question arises whether you should,

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when that attack subsides, remove the appendix, or let her run the risk consequent upon having a diseased appendix in her peritoneal cavity during the remainder of her pregnancy. Of course one's prejudice is to do nothing whatever of a surgical kind in pregnancy, to avoid all operations which you can justifiably postpone. But I am clearly of opinion that if you have evidence of disease in the appendix in the early months of pregnancy, you should remove the appendix without delay. I should much prefer to operate during a period of quiescence, that is to say while the patient is apparently quite well, but the fact of pregnancy certainly increases the urgency of the operation.

(4) *A Case of Thyroid Adenoma in a Septuagenaria.*

Here is a woman æt. 72 years, who has been sent to us for an opinion. During the last two or three years she has noticed her neck enlarging. You can easily see a tumour in front of her neck. It is ovoid in shape, and reaches down to the sternum and up to the level of the top of the thyroid cartilage on the right side. Below it occupies the space between the two sterno-mastoid muscles and lies deep to them. The anterior jugular veins are full, but the skin and superficial tissues over the swelling are normal in appearance. I ask her to swallow and you observe that the tumour moves vertically, and we are told by the student who is examining it for us that it fluctuates, and that he thinks it is a cyst connected with the thyroid body. It is easy to be sure that this is a thyroid swelling. We know that when a person swallows we can see his trachea ascend and then descend. And we know that the isthmus of the thyroid is firmly fixed to the trachea and must move with it unless it is held by some inflammatory or malignant infiltration. But not only have we this striking evidence that the swelling is fixed to the trachea but we notice that the position of the swelling, and to some extent its outline, corresponds with the position and outline of the thyroid gland. There is no difficulty therefore in being quite sure that this is a thyroid swelling, and we have next to determine the nature of the enlargement. The forms of enlargement of the thyroid body are, parenchymatous goitre, adenoma, exophthalmic goitre, carcinoma, sarcoma, acute inflammation, a little swelling of the thyroid body which sometimes occurs in health, and which we may therefore call physiological, and very rarely we have tubercle and gumma of the thyroid, and, rarer still, hydatids. Let us omit the very rare conditions, and take the others in the order of frequency. The physiological form we can dismiss as it is not of surgical importance. Then we have left over parenchymatous bronchocele, adenomatous bronchocele, pulsating or exophthalmic goitre, carcinoma and sarcoma, and acute inflammation, which is rare. This last may be found complicating typhoid fever, and it may quickly run on to abscess and may cause grave pressure symptoms on the trachea if not quickly relieved by a free incision. I do not include cystic adenoma or cystic goitre in my list, not because there is no such thing, but because in a large number of cases you are unable to tell whether a swelling in the thyroid body is a cyst or a soft adenoma. Then, again, in many cases you get the two conditions together, part of the swelling being a soft solid growth and part cystic. The origin of these two forms of disease is probably identical, and the treatment of the two is certainly identical. So in this busy life of ours it is not worth a man's while to make up his mind whether an adenoma is a soft solid adenoma or a cystic adenoma; and most experienced surgeons will tell you that they make mistakes when they try to make this distinction. So be content to be able to recognise adenoma, whether solid, fluid, or both. To return to our patient: this is a strictly unilateral swelling. A parenchymatous enlargement is one which affects the whole thyroid body, though perhaps not uniformly. As this is a tumour limited to the right half of the thyroid, it is not parenchymatous. Can you exclude exophthalmic goitre? I am told we can because there is no exophthalmos, but you must remember some cases of exophthalmic goitre have no exophthalmos. Her hand is steady when she puts it out, and her pulse does not reveal any tachycardia; its rate is 92, and you can always safely deduct a few beats when a patient is undergoing examination with a number of students round her. She has nothing which we can dignify by the term "tachycardia." You will notice that her eyelid and eyeball move synchronously, and when she looks up she wrinkles her brow. Further, the tumour does not pulsate. Do not be misled by

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thinking that a patient who has an adenoma in the thyroid cannot at the same time have Baselow's disease; the two may be associated. It is very important to remember that possible association. You may have a very unsymmetrical enlargement of the thyroid body which is associated with exophthalmos and tachycardia and the other signs which we have just mentioned. Remember now the patient's age; she is seventy-two, and that is not a common age for patients to come to us with adenoma of the thyroid; at such an age the question of malignant disease would certainly arise. The tumour has a very defined outline, and we are told it is not adherent to the skin or to the other tissues over it. There are many structures between the skin and the thyroid gland, namely, the superficial and deep fasciæ, the platysma, the sterno-mastoid, the sterno-hyoid, and the sterno-thyroid muscles, and a thin layer of fascia immediately over the thyroid gland. So a malignant tumour may infiltrate parts outside the thyroid and yet that infiltration may not have reached the skin. But the malignant infiltration first of all spreads through the thyroid body. This woman comes to us two years after she has noticed the swelling in her neck, and it is still limited to the right lobe of her thyroid body. That fact in itself is strong evidence against malignant infiltration. And not only can I lift up a fold of tissue deep down, but she swallows without any difficulty whatever, and there is no difficulty in breathing. Malignant bronchocele tends to spread into the cartilages of the trachea, but not in the first instance. It next fixes itself to the carotid sheath and binds itself to the œsophagus and the spine, and quickly interferes with comfortable deglutition. She has nothing of that kind. We need not consider acute bronchocele because she has had it two years. It is evident, therefore, that this is an adenoma of the thyroid. She tells us it is getting larger but not rapidly. Two years ago, when she first noticed it, it was the size of a cherry, and it has never troubled her. She has come to get an opinion as to whether this adenoma should be removed. I think that we should advise that it be left alone, because she is old and not at all robust, and the disease is not causing her trouble. You will notice that she limps and has lameness due to hip disease, in consequence of which she has had to give up work. If she were twenty-seven years of age instead of seventy-two one would advise her to have it removed. That is the only surgical treatment. There is no good to be done by painting it or injecting it or by giving medicines to remove it. If she had had dyspnœa I should have said that the risk from her dyspnœa would be worse than the risk from the operation and that it should be done; but under the present circumstances I am sure it is far better to leave it alone.

(5) *A Case of Cancer of the Rectum.*

Here is a single man æt 33 years, who at thirteen years of age began to be troubled with "piles." But this gave him only slight inconvenience until he reached the age of twenty-eight, and then it got worse and caused much pain at times. Three years ago the "piles" became very bad, and he at that time began to bleed a little after a motion was passed. But he has sought no advice until ten days ago, when about half a teacupful of blood passed with the motions. At this time the patient's attention was directed to the fact that the motions were small in diameter, and that a little mucus was passed with them. Thus, till ten days ago there appears to have been nothing sufficient to attract his attention, or to cause him to seek medical advice, except a very little hæmorrhage after stool. He says that passing a hard motion gave pain and was followed by bleeding, but a natural soft motion was painless. He has suffered much from constipation. His life has been an active one, out of doors, and he tells us that the day before he came into the hospital he walked twenty miles. Before making a rectal examination let us examine the abdomen and notice four points, (1) whether there is any evidence of distension of any part of the intestine; (2) whether there is visible peristalsis indicating obstruction to the onward passage of the intestinal contents; (3) whether the liver is enlarged; and (4) the state of the inguinal, iliac, and lumbar glands. The result on all these points is negative. We notice further that he is a well-nourished man, of good colour, not anæmic, and with a sunburnt face. There is a slight fur on the tongue, but it is only trifling. On looking at him you would say he is a healthy, strong, robust young man. I shall now ask one of you to examine the rectum carefully and to give us a good description of what he finds. He must take care to get the patient into

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a good position, lying well over on his left side with the buttocks on the edge of the bed, the legs drawn well up, and with the left arm under him, so that he cannot move. First look to see whether there is fistula or pile, or any hæmorrhage or swelling. Then insert a well-lubricated finger very gently and slowly, and note the degree of resistance which the finger meets with, whether there is unusual resistance, or whether the sphincter is patulous and soft. Also notice whether the examination seems to cause him undue pain. We are told that there is a growth on the anterior part of the bowel which reaches from just above the anus, for two inches higher up; the finger can be passed above it. The posterior part of the bowel is free; the growth is firm and uneven. It is thought to be fixed, but as it is on the anterior part of the bowel the adhesion would be to the bladder or prostate, and we cannot be so sure of fixture to these parts as to the sacrum, because they are themselves movable. The fact that the growth moves a little is not a proof that it moves over the prostate and bladder. There is no other evidence that the growth has infiltrated the muscular tissue of the bladder and prostate, for he has no pain when he passes water. We want to know particularly whether we have here an infiltrating growth. That raises a very interesting point. What is the great characteristic by which a non-infiltrating differs from an infiltrating growth? What happens to a non-infiltrating growth of the rectum as it grows? It drops into the lumen of the bowel and it is then called a "polypus." In other words, a "polypus" is the expression in terms of the rectum of the fact of a non-infiltrating growth. For we get the same fact expressed in different ways in different parts of the body. But in the case of an infiltration, whatever its nature, you find it spreads along the bowel, tending to become circular and then tending to grow up to the bowel and become tubular. In this case there is nothing polypoid, nothing which you can put your finger round in making the examination; but you feel that a certain part of the circumference of the bowel is firm, thickened, warty, ulcerated, and that is evidence of its being malignant. And more than that: if you pass your finger to the centre of the growth, you notice a deep ulcer; it has actually eaten well into the wall of the bowel; and yet at the floor of it is the warty, thick, infiltrating growth. There is still the same kind of base, extending into the wall of the bowel. It is therefore an infiltrating ulcerating growth. I agree with the diagnosis that this man has columnar-celled carcinoma of the rectum. So far as the physical diagnosis goes, with the finger in the rectum there are no difficulties at all. The striking thing about the case, which I want you to remember well, is the lack of indication in the general condition of the patient of the presence of grave disease. He is in excellent general health, and you have heard him say that he walked twenty miles some days ago; he has been doing his ordinary out-door work, and shows no sign of constitutional deterioration; his physical vigour is as good as that of anyone here. But more striking still is the fact that he has had practically no pain, and only a most trifling hæmorrhage—that is to say, last week a half-teacupful. He has not had any great discharge of mucus, nor frequent calls to stool, nor any of the symptoms which you read of in your text-books as characterising the early stages of carcinoma of the rectum. There has been a little occasional oozing of blood, and pain when he strains at stool, but that is what many have when passing a hard motion. There has been scarcely anything in his case pointing to symptoms of grave disease, and it was not until the doctor he went to see insisted upon a local examination that he could determine there was anything serious the matter. The moral of that is to carefully examine every patient who comes to you complaining of "piles" or of any such symptom. Never prescribe without such examination. It would be as if a person came complaining of difficulty of sight, and the ophthalmic surgeon wrote a prescription for a pill or powder without examining the eye. A man who complains of "piles" may have a fistula, or piles, or a fissure, or carcinoma, and it is not safe or right to attempt to treat such cases without examination of the rectum. You will not have a better case than this to demonstrate the absolute necessity of physical examination in every case which comes before you, nor one which more forcibly illustrates the extremely slight symptoms which you may get with quite grave disease. And that has another very important bearing. Every now and then we are called upon to deal with a case of quite abrupt intestinal obstruction, and in which you can get no history from the patient of anything having been previously wrong with him. He will say that up

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to a certain day he had been having regular motions, though possibly he had a little constipation, and that on that particular date his motions stopped, there was then swelling of the abdomen, and he became seriously ill. On examining him you find the abdomen distended and complete intestinal obstruction. You are naturally inclined to think that the man cannot be suffering from a *chronic* disease, but as a matter of fact that is precisely the case. Over and over again we find that chronic obstruction goes on without any symptoms at all, just as in this man's case, until some "accidental" condition sets up complete intestinal obstruction. The most striking case I ever had of it was that of a man who came into this ward with a greatly distended abdomen; it was as tight as a drum, and I could make out nothing about it, except that there was complete obstruction; neither motion nor flatus had passed for several days. So distended was he that no useful examination of the abdomen at all could be made: one could not even tell whether one part of his colon was more distended than another. I made an incision in the middle line because I did not know where the obstruction was, and I found it in the sigmoid flexure. I then made a second wound on the left, drew out the bowel, fixed it to the skin and opened it, and subsequently excised the growth. On examining it I found an annular growth of the sigmoid flexure of the colon; the lumen was so small that an orange-pip had blocked it up like a cork in a bottle, and this had brought about complete obstruction. We cross-examined the man afterwards as to his history, and he assured us that up to the very day of the onset of the acute symptoms he had had regular motions every day, that there had been no "spurious diarrhœa," nor diarrhœa alternating with constipation, nor any passage of blood or of mucus such as the text-books mention. That is an extreme case; but such a case as we have before us now helps us to understand it, for here is a growth quite close to the sensitive anal canal, but he has had almost no pain and only trifling hæmorrhage.

What shall we do for this man? Ought we to excise this growth or to do a colotomy? Excision of the rectum is certainly one of our good operations, which often gives permanent results, though not in every case. Therefore always let your first thought in the treatment of any case of malignant disease of the rectum be, Can I excise it? It is not until you have satisfied yourself that it is unsuitable for excision that you turn about for another method of affording relief to the patient. What are the particular points which we have to attend to before we advise excision of the rectum? The first is, can we, with a reasonable operation, remove all the obvious disease? And, remember, we can never leave out that word "obvious" in connection with malignant disease, because there may be an infection of glands and other parts which are beyond the reach of our examination. To answer this question we must know first whether the growth has extended beyond the wall of the bowel to the parts around, either to the sacrum behind or to the prostate and bladder or uterus in front. Next we must know whether there are any secondary growths in parts beyond the rectum altogether, such as the glands in the hollow of the sacrum, or in the groin or the liver. It is important also to know the length of the bowel which it will be necessary to remove, although this does not enter into the question so much as it used to. Formerly disease which was beyond the reach of the examining finger was beyond the reach of operation. Now we are not afraid of removing almost any length of the rectum, though, of course, the length of the bowel removed adds to the severity of the operation. The other great question we must put to ourselves is whether there is any condition in the patient which would render the operation of excision of the rectum an improper one to perform. Applying all this to the patient before us, the disease is apparently limited to the rectum itself. I am hopeful that there is no extension to the bladder or prostate. I cannot speak quite confidently about that, however. The liver is not enlarged, there are no enlarged glands in the groin, nor can I feel any in the pelvis, and with regard to the length of the bowel to be removed, the cancer is low down, and the finger can be passed above it. As to his general condition, we have already noticed his robust appearance and good muscles, there is nothing abnormal in his urine, and his heart is sound, and we therefore decide that the case is a suitable one for operation. What route shall we choose in this case? You rightly say the perineal one. And what cautions will you give me in doing the operation? Why, to be sure, you will urge me, whatever else I do, to take great care to go well

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beyond the disease, and not to wound the urethra and bladder. To avoid the urethra I shall have a bougie in it during the operation, to enable the contour of it to be felt. Shaving off a little of the prostate would not matter much. But if I find that the malignant infiltration has extended through the rectum into the wall of the bladder I shall stop the operation. Do not think you are bound to complete an operation because you have started it, if you meet with unforeseen conditions; that way lies disaster. Another piece of advice I should expect to receive from you would be to keep the malignant growth away from the wound, lest it become infected, for it seems to be possible to implant carcinoma in a wound. The operation is completed by fastening the bowel to the skin by sutures, which not only include the skin and all the thickness of the bowel, but pass deeply in the wall of the wound, and as far as possible obliterate the cavity between the bowel and the skin. Ten years ago the first thing you would have told me would have been not to wound the peritoneum. Now we make no mention of that. If it is necessary to open the peritoneum we deliberately do it, and sometimes we close the cavity by sutures, sometimes we do not, but the one thing we chiefly concentrate our attention upon is the complete and wide removal of the infiltrated and infected tissues. In this case, then, we recommend the operation with a confident expectation of the patient's recovery, and with a good hope that he will be permanently freed from his disease.

[*N.B.*—The operation was performed four days later, and the patient made a good recovery.]

October 8th, 1906.

Reibold says that so far four cases of what appears with reasonable certainty to have been tuberculous meningitis have been reported, in which recovery followed. To these he adds a fifth, in which he thinks the favourable outcome was brought about by persistent resort to spinal puncture. The patient was a girl of sixteen years, and in all 574 c.c. of cerebrospinal fluid were withdrawn at 24 punctures, which at first were repeated at daily intervals and later on less frequently. The author believes in the beneficial effect of the measure, and urges that it be more frequently resorted to.—*Medical Record*, vol. lxx, No. 12.

The Channels of Infection in Tuberculosis.

By Hugh Walsham, M.A., M.D., F.R.C.P., Physician to Out-patients City of London Hospital for Diseases of the Chest. London: Bale, Son & Danielsson.

There are no more important factors in the ætiology of tuberculosis than the determination of the primary seat of the disease and the channels of infection. Dr. Hugh Walsham has made a valuable series of observations on these points, and these observations he has worked up into the essay to which the Weber-Parkes Prize was awarded in 1903. The essay was accompanied by 280 microscopical preparations, which show the large amount of original work which Dr. Walsham has performed. The essay consists of two parts. In the first part Dr. Walsham deals fully and systematically with the various channels of infection in tuberculosis. These he considers are five in number, namely: (1) Hereditary transmission; (2) The lymphatic vessels; (3) The blood-vessels; (4) Epithelial channels; and (5) Inoculation into the skin or tissues.

Dr. Walsham believes that the tubercle bacillus can be passed through a normal intact mucous membrane without leaving any trace behind it. The importance of the tonsil as an infection atrium in tuberculosis is fully insisted on. The tonsil may or may not suffer in the passage of tubercle bacilli through it. The consideration of primary tuberculosis of the tonsil forms one of the best sections in the book.

In part II. Dr. Walsham deals with the conditions, original or acquired, which render the tissues vulnerable. He considers that "of all the various conditions which lower resistance and render the tissues vulnerable, two stand out in marked contrast to the rest. They deserve special attention because they are the commonest and, therefore, the most important causes which render the tissues or organs of the body liable to the attack of the tubercle bacillus. They are: (1) The want of fresh air and sunlight, and (2) insufficient food. These two conditions act as general rather than local causes." He then discusses the relationship of alcohol, diabetes, cardiac diseases, lymphadenoma, pregnancy and other diseases to tuberculosis. A useful bibliography concludes this interesting volume, which should be perused by all who are interested in the subject of the channels of infection in tuberculosis.

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